South African Medical Journal
Suid-Afrikaanse Tydskrif vir Geneeskunde

Vol. 24, No. 16 Cape Town, 22 April 1950 Weekly 2s

HYPERINSULINISM OR HYPOGLYCAEMIA DUE TO AN ISLET CELL TUMOUR

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This interesting condition was first discovered by Searle Harris in 1924, and in 1929 the first successful removal of an islet cell tumour from the pancreas with relief of hypoglycaemia, was reported.

The symptoms associated with this condition may be divided into 3 groups:—

1. Disturbances of the autonomic system, such as nausea, vomiting, dizziness, pallor and sweating.
2. Disturbance of the central nervous system, such as convulsions and tonic and clonic spasms.
3. Psychic disturbances, such as amnesia, confusion, mania and coma.

Our case showed, at different times during observation, symptoms in each of these groups.

Pathology. The commonest sites for these tumours is the body and tail of the pancreas where they usually occur on the surface or in the substance of the gland. More rarely they are found in the head. They are small and vary from a few mm. in diameter to about 2 centimetres and are greyish or reddish-grey in colour and are frequently non-encapsulated. They may be multiple, when usually only one produces insulin. The tumour in our case was found on the postero inferior surface of the tail. Dr. Murray of S.A.I.M.R. reports:—

'Sections of this tumour from the pancreas show the structure of a nesidioblastoma. There is a general tendency to alveolar arrangement of the cells but in many areas the tumour has a papillary appearance. Mitotic activity is present and tumour tissue merges gradually with the surrounding pancreatic tissue. On the other hand the neoplastic cells are uniform in appearance and, as is usual in these tumours, it is not possible on histological grounds to determine whether the tumour is benign or malignant.'

Miss M., aged 16 years, was noticed to be unduly tired in the early mornings and in the evenings by her parents in June 1949. After breakfast she was normal and led an active life on the farm and no real notice was taken of her fatigue. At the end of September, her friends at hostel found it difficult to waken her one morning, but after shaking her for a time, they roused her and she staggered into breakfast. After this meal she returned to normal and attended school that day. At about this time they noticed that she was excessively weary and dull about supper time and again became normal after the meal. A week later she was again 'sleepy' in the morning and her friends were unable to wake her. A doctor was called in, and as she was apyrexial and no abnormal signs could be elicited, he gave her some calcium gluconate and coramine intravenously, when she immediately recovered consciousness and was able to continue at school. This happened twice more at approximately weekly intervals, with the same result.

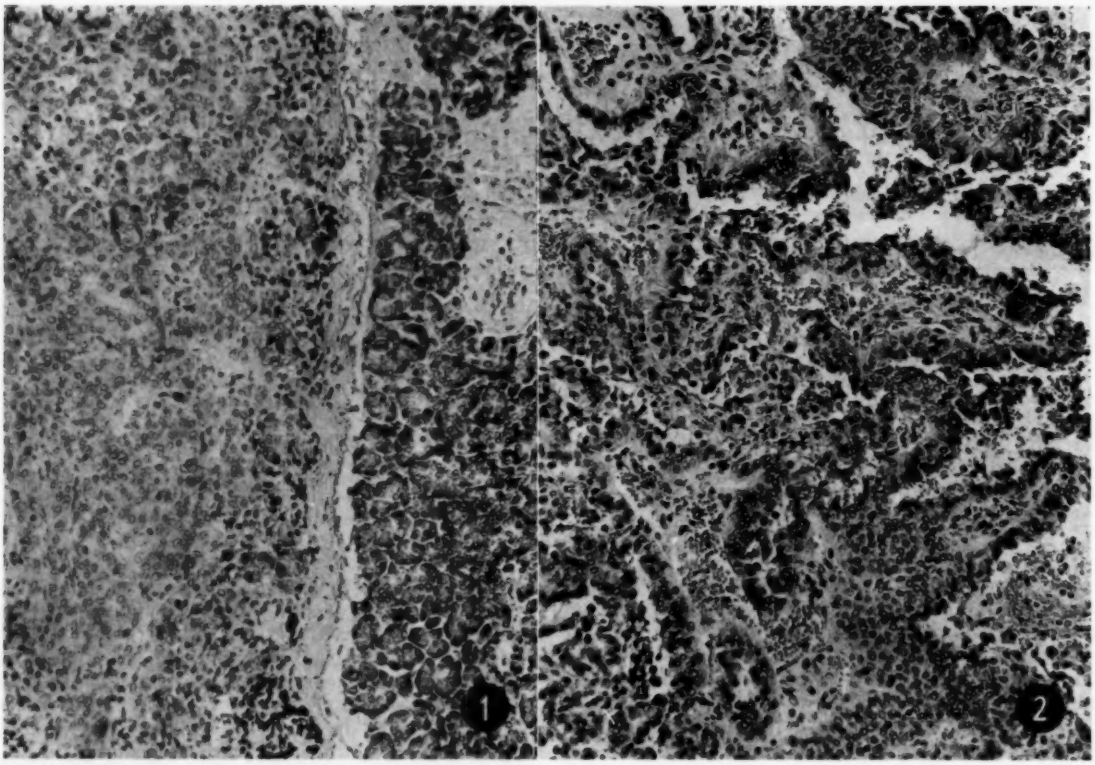
On 21 October 1949 she again became comatose, but this time she did not respond to calcium gluconate and coramine intravenously. Physical signs were of no significance—pupils were dilated and she was sweating, skin blotchy, breathing stertorous and she remained in a coma until 24 October, when she was referred to us.

Examination. A well nourished young female, slightly cyanosed, breathing stertorously, pupils dilated and tendon reflexes, other than Babinski on the left, could not be elicited. Fundi were normal and eyeballs seemed of normal tonus.

The following investigations were undertaken:—

Blood sugar and blood urea and examination of urine. While I was getting ready to do a lumbar puncture, she had an epileptiform seizure, but there was no cry and no incontinence of urine. Lumbar puncture was undertaken while she was in this fit. The fluid was clear and not under pressure.

Spinal Fluid Examination. No cells; chlorides and protein normal; sugar reduced to 50 mg. per 100 ml. Blood sugar 40 mg. per 100 ml. Blood urea 22.6 mg. per 100 ml. Urine normal. At this stage a tentative diagnosis of essential hypoglycaemia was made and she was given 50 c.c. of 50% glucose intravenously and a



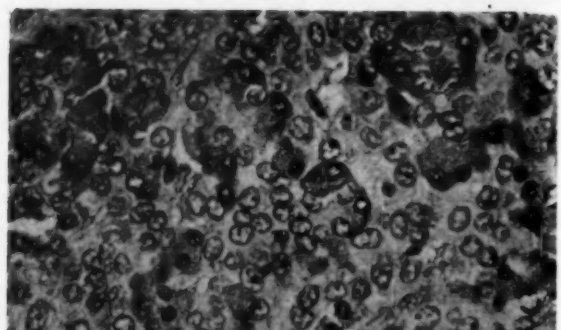


Fig. 1. Photomicrograph to show alveolar masses of neoplastic cells separated at this point from the adjacent pancreatic tissue by a thin septum of connective tissue. H. & E. x 190.

Fig. 2. Area in which the neoplasm shows a pseudo-papillary and pseudo-acinar arrangement of the cells. H. & E. x 190.

Fig. 3. High-power magnification to show infiltration of the pancreatic tissue by the neoplastic cells.

dextrose and water drip was put up. As she did not regain consciousness, we gave her 1 c.c. of pituitrin and 10 m. of adrenalin, but apart from pushing her pulse rate up, it had no effect.

At this stage it was decided to give her protein hydrolysate intravenously, and halfway through the second 500 c.c. she became conscious and her blood sugar rose to 50 mg. per 100 ml.

On 26 October she was co-operative. Intravenous drips were discontinued and she was put on to a high protein, high vitamin diet plus 1 pound of boiled sweets *per diem* and kept at rest. During her stage of coma, she developed a right basal pneumonia which responded promptly to penicillin therapy.

Repeated blood sugar examination gave consistently low readings, 40 to 50 mg. per 100 ml. A glucose tolerance test gave the following results:—

Fasting blood sugar 42 mg.: ½ hour after glucose 174 mg.; one hour, 218 mg.; 3rd., 104 mg.; 4th, 70 mg. From this stage onwards her blood sugar remained at 50 mg. per 100 ml. on the above diet and at rest.

We decided that laparotomy should be performed. An attempt was to be made to find an islet cell tumour

and, in the absence of this macroscopically, to do a partial pancreatectomy. Should symptoms recur thereafter, we contemplated a subsequent operation to remove the head of the gland.

The day before operation (7 November 1949) her fasting blood sugar rose to 60 mg. On 8 November 1949, having again put up a dextrose and water drip, operation under thiopentone, curare, oxygen and cyclopropane was undertaken through a right paramedian incision. The lesser sac was opened through the gastro-colic omentum, the pancreas identified and palpated. No tumour was found in the body or neck. The duodenum was mobilized after first wiping down the hepatic flexure, and the head of the pancreas palpated between finger and thumb, again with negative results. Now the gastro-colic omentum was ligated off for about 3 inches at the pyloric end of the stomach, the superior mesenteric vessels were identified and avoided and we began to mobilize the body of the pancreas. At this stage there was a little bleeding from vessels adjoining the splenic vein. As we approached the tail, we felt a small area on the the posterior surface of the tail, abutting against the spleen, which was of different consistency from the rest of the gland. Gently, this, together with the tail was mobilized, and there came into view a tumour the size of a small grape, dusky red and congested looking. It had no definite capsule and so it was removed together with a small portion of normal pancreas. Haemostasis was effected by mattress sutures put through the pancreas. The hole in the omentum was closed with divided sutures and a small drain was put into the lesser sac and the abdomen closed.

Post-operative findings:—

9 November: The patient regained consciouness late in the evening of 8 November, and on 9 November was very drowsy; bowel sounds had returned, no vomiting, and the drip was discontinued as the patient was now co-operative. Blood sugar, 117 mg. per 100 ml. and in the early afternoon glycosuria developed, thus showing that, in the presence of an actively secreting tumour, the normal islets were inactive, and that there is a time lag before they take up their normal functions.

10 November: Still drowsy, taking fluids well, bowels acted, no vomiting. Glycosuria disappeared, i.e. 40 hours after operation.

11 November: Still drowsy and complaining of a great deal of pain in the wound.

12 November: More wakeful, blood sugar 97 mg. per 100 ml.

13 November: More co-operative, tube removed, urine normal.

14 November: Patient better, less drowsy.

16 November: Remarkable change in mental condition, patient alert and interested in surroundings, appetite has returned. Fasting blood sugar 90 mg. per 100 ml.

18 November: Patient well. Fasting blood sugar 82 mg. per 100 ml.

19 November: Stitches removed, wound soundly healed.

20 November: Fasting blood sugar 80 mg. per 100 ml.; 2 hours after meal, blood sugar 100 mg. per 100 ml.

14 March 1950: Fasting blood sugar, 72 mg. per 100 ml. Patient well.

DISCUSSION

Other causes of hypoglycaemia:—

(*a*) Low carbohydrate intake with high output, e.g., exercise or low renal threshold for sugar.

(*b*) Liver diseases, inflammatory or neoplastic, where 80% of functional liver is destroyed.

(*c*) Distant endocrine disturbances, e.g., pituitary and hypothalamic diseases like Simmonds' disease and occasionally in toxic goitre.

These were eliminated by the fact that this patient's diet was not lacking in carbohydrates before her attacks, and that her urine contained no sugar. There was no evidence of hepatitis or other liver disease. Distant endocrine disturbances were excluded by examination of the thyroid which was normal, and there were no signs of Graves' disease. X-ray of the skull was normal and spinal fluid findings, pressure and chemistry were normal. Thus the diagnosis of islet cell tumour as the cause of her condition was established.

Having come to this conclusion, we decided that operation, in spite of the 16% mortality associated with this procedure, was justified because of the increasing frequency and severity of the attacks of hypoglycaemia our patient was having, culminating in this final attack where she was in coma for a week and from the fourth day onwards was having epileptiform seizures and that, in spite of complete rest and adequate diet, her blood sugar remained at the low limit of 50 mg. per 100 ml. We believe that in those cases where the hypoglycaemia is relieved by sugar quickly, and where the fasting blood sugar can be brought to normal limits by adequate intake of food, operative measures should not be undertaken.

SUMMARY

1. A case of hypoglycaemia due to a nesidioblastoma has been described together with the operative technique for the removal of the tumour.

2. The pathology of this tumour together with accompanying photomicrographs has been shown.

3. The post-operative changes, both mental and physical, have been recorded.

Our gratitude is due to Dr. Murray of the S.A.I.M.R. for the pathological report and photomicrographs; to Dr. N. Downes, our anaesthetist; and to Drs. Tandy and Stephens for their help and encouragement in compiling this paper.

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ABSTRACT

Debray, J. R. (1949): *Treatment of Gonorrhoea in Women by Local Submucous Injection of Penicillin.* Presse Médicale, **57,** 1134.

This technique was developed as a result of the observation that the standard treatment of gonorrhoea by penicillin was much less effective in women than in men.

Using a fine dental needle, six to eight submucous injections of 1.5 c.c. penicillin solution (500,000 U in 20 c.c. saline) are made around the cervical canal. Four injections are made round the urethra, a surface anaesthetic being used previously. If there is evidence of infection of Skene's or Bartholin's glands, local injections are made.

In the first instance treatment is given every other day for four visits, but it may be continued if the results are not then satisfactory; 400,000 U penicillin in aqueous solution (in two doses, morning and evening) are also given intramuscularly on the first and last days of treatment, and thiazomide 3 gm. daily for three days at the start. Results are controlled by cultures and have been markedly better than with standard treatment alone.

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VAN DIE REDAKSIE

DIE STATUS VAN DIE INTERN

Met die aanname van die Wysigingswetsontwerp op Geneeshere, Tandartse en Aptekers deur die Senaat in Maart 1950 is 'n nuwe status vir die gegradueerde geskep wat onlangs in geneeskunde gepromoveer het. Hy is nou nie meer 'n student in die sin waarin hy geregistreer het toe hy die tweede jaar van die geneeskundige leerplan begin het nie; hy is ook nie 'n geregistreerde geneesheer soos in artikel 22 (3) van die ongewysigde Wet op Geneeshere, Tandartse en Aptekers beoog is nie, waar 'n poging aangewend is om hom as 'n ten volle geregistreerde geneesheer te erken wat by die beoefening van sy beroep beperk sou word tot die goedgekeurde inrigting waarby hy vir 12 maande aangestel is.

Daar sal in die algemeen saamgestem word dat wat die Mediese Vereniging, die mediese beroep sowel as die Geneeskundige Raad in gedagte gehad het, die waarborg van die betrekkings was van inwonende geneeshere nadat hulle gegradueer het sodat aan pas gegradueerdes die geleentheid gegee kan word om geleidelik verantwoordelikheid te aanvaar. Die oogmerk was nie om die kursus met een jaar te verleng en die intern in 'n mediese student om te skep wat in sy sewende jaar is nie. Dit was almal se begeerte dat omstandighede geskep word waarin 'n ten volle geregistreerde geneesheer vir een jaar sy praktyk tot 'n goedgekeurde inrigting sou beperk.

As gevolg van een van daardie onverklaarbare lotgevalle wat die gebruik van woorde te beurt kan val, het die 1950-wysiging van die Wet op Geneeshere, Tandartse en Aptekers 'n status vir die intern geskep wat presies die teenoorgestelde is as wat bedoel is. Dit kom nou daarop neer dat hy 'n mediese student in sy sewende jaar is wat deur middel van 'n spesiale statutêre vrystelling voorskrifte vir gewoontevormende middels kan uitskryf maar nie 'n doodsertifikaat kan onderteken, 'n siektesertifikaat vir lede van die staatsdiens kan uitreik of kranksinniges kan sertifiseer nie.

Sy verhouding teenoor die verpleegpersoneel van die hospitaal waarin hy sy sewende jaar van opleiding ontvang, word klaarblyklik ondermyn aangesien hy nie die status van 'n geregistreerde geneesheer het nie. Dit is ook netso waarskynlik dat hy nie op die gebruik van die titel „dokter" geregtig sal wees of homself as 'n geneesheer sal kan beskryf nie.

Sekere moeilike wetlike gevolge mag voortspruit uit die veranderde status van die gegradueerde wat pas in geneeskunde gepromoveer het. Dit is onwaarskynlik dat ons howe sy getuienis as dié van 'n vakkundige sal aanvaar en dit sal klaarblyklik 'n uiters onbevredigende toedrag van sake in verband met die talryke aanrandings- en ander gevalle skep wat daagliks in ons

EDITORIAL

THE STATUS OF THE INTERN

With the passage of the Medical, Dental and Pharmacy Amendment Bill through the Senate in March 1950, a new status has been created for the recent medical graduate. He is now not a student in the sense in which he registered when he entered his second year of the medical curriculum; nor is he a registered medical practitioner such as was envisaged for him in terms of section 22 (3) of the unamended Medical, Dental and Pharmacy Act, where an attempt was made to recognize him as a fully registered practitioner who would be limited in the excercise of his calling to the approved institution to which he had been appointed for 12 months.

It will be agreed generally that what the Medical Association, the medical profession as well as the Medical Council had in mind was the guarantee of posts for all house-men after graduation so as to give recent graduates opportunities for the graduated assumption of responsibility. The object was not to lengthen the course by one year and to convert the intern into a seventh year medical student. The desire which all shared in common was to create a situation in which a fully registered medical practitioner would restrict his practice to an approved institution for one year.

By one of those inexplicable adventures that can befall the use of words, the 1950 amendment of the Medical, Dental and Pharmacy Act created a status for the intern exactly the opposite of what was intended. He is now, in effect, a seventh year medical student who by a special statutory dispensation can write prescriptions for habit-forming drugs, but who cannot sign a death certificate, or issue a sickness certificate for members of the public service, or certify the insane.

His position *vis-a-vis* the nursing staff of the hospital in which he is undergoing his seventh year of training, obviously becomes undermined as he has not got the status of a registered medical practitioner. It is equally likely that he will not be entitled to the use of the prefix 'Dr.' or to describe himself as a medical practitioner.

Certain difficult legal consequences may flow from the altered status of the recent medical graduate. It is unlikely that our Courts will receive his testimony as that of an expert witness, and this will obviously create a very unsatisfactory state of affairs in connexion with the numerous assault and other cases which are heard daily in our Courts through the length

howe deur die lengte en breedte van die land verhoor word. Die waarde van sy getuienis kan nie hoër geskat word nie as dié van 'n mediese student nie en aangesien die intern by kritieke stadiums van die behandeling van die aangerande en aangeklaagde persone betrokke was, skyn dit onbehoorlik om hom sy reg te ontneem om daardie wetlike verantwoordelikheid vir sy handelinge te aanvaar wat hy tot dusver geniet het.

Dit is ook twyfelagtig of die intern behalwe onder toesig 'n narkose kan toedien en sekere lastige probleme mag in die geval van 'n sterfgeval onder narkose uit die aard van die verantwoordelikheid voortspruit.

Daar kan nouliks 'n onoorkomelike moeilikheid wees in verband met die wysiging van die Wet ten einde die intern te maak wat almal hom bedoel het om te wees, nl. 'n ten volle geregistreerde geneesheer wie se bedrywighede vir 12 maande beperk word tot die goedgekeurde inrigting waarby hy aangestel is. Die Wet self verskaf presedente vir hierdie soort beperkte praktyk en ons is seker dat die Geneeskundige Raad hom by die Mediese Vereniging sal skaar in 'n ernstige begeerte vir die verbetering van wat as een van daardie ongelukke in die omstandighede van woorde beskou moet word wat, soos bekend is, die moeilike probleme van wetsopstelling ingewikkelder maak.

DIE TERUGWERKENDE AARD VAN VERPLIGTE INTERNSKAP

Dit is bes moontlik dat die Wet op Geneeshere, Tandartse en Aptekers op die drempel van 'n kroniese toestand van wysiging staan; maar hierdie onsekere toestand van sommige beginsels van die Wet moet beskou word as die onvermydelike gevolge van die heraanpassings wat met kenteringstye gepaard gaan.

Indien 'n poging aangewend gaan word om die Wet ten opsigte van die status van die intern te wysig, moet die aandag weereens gevestig word op die onwenslikheid om internskap vir diegene verpligtend te maak wat reeds 'n aanvang met die studiekursus gemaak het voor die afkondiging van die regulasies van 1948, regulasies wat destyds deur mense wat die onderwerp ernstig bestudeer as ongeldig beskou is. Hierdie probleem is voorheen reeds in ons redaksiekolomme bespreek[1].

Die wysiging van 1950 is bedoel om die regulasies geldig te maak wat *ultra vires* ten opsigte van die Wet beskou is. Dit is egter twyfelagtig of die regulasies in verband met verpligte internskap wettiglik van terugwerkende krag gemaak kan word, selfs ten spyte van die onlangse wysiging van die Wet. Soos kol. Pilkington Jordan gedurende die Komiteestadium van die debat oor die Wysigingswetsontwerp op Geneeshere, Tandartse en Aptekers[2] aangedui het, *gee die magtigende bepaling in die* 1946*-wysiging van die Wet nêrens te kenne en kan dit geensins vertolk word as wou dit te kenne gee dat hierdie magtigingsgesag bedoel was om enige terugwerkende krag te veroorloof nie.* Indien hierdie mening juis is (en dit is moeilik te begrype hoe hierdie vertolking betwis kan word) dan word 'n paar

1. Hierdie *Tydskrif*, 11 September 1948, bl. 541, en 11 Maart 1950, bl. 166.
2. Debatte van die Volksraad (Hansard), No. 5, 20 Februarie 1950, kolom 1709.

and the breadth of the land. The value of his evidence can be rated no higher than that of a medical student and as the intern is likely to have been involved at critical stages in the treatment of assaulted and accused persons, it seems improper to deprive him of the right to assume that full legal responsibility which he has enjoyed until now for the acts he has performed.

It is also doubtful whether the intern can administer an anaesthetic except under supervision, and certain awkward problems may arise about the nature of the responsibility in the case of anaesthetic deaths.

There can hardly be any insuperable difficulty about amending the Act so as to make the intern what everyone intended him to be, viz. a fully registered medical practitioner whose activities are restricted for 12 months to the approved institution to which he has been appointed. The Act itself provides precedents for this type of restricted practice, and we feel sure that the Medical Council itself will join with the Medical Association in an earnest desire to revise what must be regarded as one of those accidents of verbal circumstance not unknown to complicate the difficult problems of legal draftmanship.

THE RETROSPECTIVE NATURE OF COMPULSORY INTERNSHIP

It is quite possible that the Medical, Dental and Pharmacy Act is about to pass into a state of chronic amendment; but this precarious condition of certain principles of the Act should be viewed as the necessary consequence of the readjustments appropriate to changing times.

If an attempt is to be made to amend the Act in respect of the status of the intern, it is once again appropriate to draw attention to the undesirability of making internship compulsory for those who had already embarked upon their course of study before the promulagtion of the 1948 regulations, regulations which serious students of the subject regarded as invalid at the time. This problem has been discussed in our Editorial columns before[1].

The 1950 amendment to the Act purports to validate regulations which have been regarded as *ultra vires* the Act. It is doubtful, however, whether, even in spite of the recent amendment to the Act, the regulations for compulsory internship can lawfully be made retrospective. As Col. Pilkington Jordan pointed out during the Committee stage of the debate on the Medical, Dental and Pharmacy Amendment Bill[2], *the enabling provision in the* 1946 *amendment to the Act nowhere indicates and in no manner can be construed to indicate that this enabling authority was intended to permit any retrospective effect.* If this view is correct (and it is difficult to see how this inter-

1. This *Journal*, 11 September 1948, p. 541; and 11 March 1950, p. 166.
2. House of Assembly Debates (Hansard), No. 5, 20 February 1950, p. 1657.

honderd gegradueerdes hulle volle wetlike status as geregistreerde geneeshere ontneem en enige ondernemende gegradueerde kan sy regte in die Hooggeregshof toets. Hoe dit ook al sy, omdat die regulasies van 1948, of hulle geldig was al dan nie, die voorneme om verpligte internskap in te voer aan die wêreld te kenne gegee het, sal dit onredelik wees om die terugwerkende krag van die wetgewing verder as hierdie datum terug te stel. Dit is belangrik dat hierdie eenvoudige beginsels van geregtigheid en die klaarblyklike morele verpligting erken word dat studente wat reeds voor die end van 1948 vir die mediese leerplan geregistreer het, nie gestrem moet word nie.

DIE TOEREIKENDHEID VAN INTERNBETREKKINGS

Reeds in 1948 het die Federale Raad van hierdie Vereniging sy besorgdheid daaroor uitgespreek dat verpligte internskap 'n intern mag dwing om 'n jaar lank na-graadse opleiding in hospitale te ontvang waar toestande nie van erkende opvoedkundige waarde is nie[3]. Dit blyk nou dat daar van interns vereis mag word om hulle jaar van internskap in gestigte vir sielsiekes. by gesondheidsentrums of selfs as leerlinge by geneeshere deur te bring. Dit is heeltemal onnodig om dit te bespreek of hierdie drie klasse genoegsame geleentheid verskaf vir die nodige onderrig in die geleidelike aanvaarding van verantwoordelikheid ten opsigte van die probleme van geneeskundige praktyk. Bloot die feit dat hierdie drie klasse van internskap aansienlike bespreking oor hulle geskiktheid uitlok, behoort genoeg te wees om die gevare te beklemtoon wat daarin skuil dat die interns soos Prokrustes se slagoffers gerek word om aan die regulasies te voldoen. Bloot die feit dat aanstellings by gesondheidsentrums en as leerlinge by geneeshere iets geword het wat ernstig oorweeg moet word, verleen krag aan die mening dat die instelling van die regulasies in sake verpligte internskap voorbarig was. Alhoewel die Wysigingswetsontwerp deur albei huise van die Parlement aangeneem is en dit afgekondig is, is daar veel daarvoor te sê dat die toepassing daarvan vertraag word totdat hierdie ernstige gebreke van die toestand verhelp kan word.

3. Hierdie *Tydskrif*, 11 September 1948, bl. 542.

pretation can be challenged) then several hundred graduates are being improperly deprived of their full legal status as registered medical practitioners, and any enterprising graduate could test his rights in the Supreme Court. However, because the 1948 regulations, whether valid or not, did actually give notice to the world of the intention to introduce compulsory internship, it would be unreasonable to stretch the retrospective effect of the legislation beyond this date. It is important to recognize these simple principles of justice and the obvious moral obligation not to handicap students who had already registered for the medical curriculum before the end of 1948.

THE ADEQUACY OF THE INTERN POSTS

As far back as 1948, the Federal Council of this Association expressed its concern lest compulsory internship compel the intern to do a year of post-graduate training in hospitals where conditions might not be of acceptable educational value[3]. It now appears that interns may be required to spend their intern year in Mental Hospitals, in Health Centres or even as apprentices to general practitioners.

It is palpably unnecessary to debate whether these three categories provide appropriate ways in which interns may receive their necessary instruction in the graduated assumption of responsibility for the problems of medical practice. The mere fact that these three classes of internship raise considerable debate about their suitability should be sufficient to emphasize the dangers of a Procrustean stretching of the intern to fit the regulations. The very fact that appointment to Health Centres or apprenticeship to general practitioners has become something which must seriously be entertained, strongly supports the view that the introduction of the compulsory internship regulations has been premature. Although the amending Bill has passed through both Houses of Parliament and has been promulgated, there is much to be said for delaying its implementation until such time as these serious defects in the situation can be remedied.

3. This *Journal*, 11 September 1948, p. 542.

FIBROCYSTIC DISEASE OF THE PANCREAS

REPORT OF A CASE

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This disease of infants and children has also been called fibrosis of the pancreas, cystic fibrosis of the pancreas, congenital steatorrhoea and congenital pancreatic deficiency.

The fibrotic lesion in the pancreas was first described by Landsteiner[1] in 1905, and later a few reports associated it with steatorrhoea. It was not until 1938 that the clear description of the disease by Harper[2], Andersen[3], Blackfan and May[4] established it as a clinical entity. Since then numerous cases have been reported in the literature. No such record has been found in the medical literature of South Africa. It is apparent that cases occur more frequently than is realized and, if the condition is kept in mind, many more infants with the disease will be discovered. Previously, cases have been diagnosed as coeliac disease, idiopathic steatorrhoea,

chronic bronchitis, bronchopneumonia or bronchiectasis.

Clinically the disease is characterized by steatorrhoea at an early age of infancy; a failure to gain weight despite a normal diet and an excellent appetite, some lung infection and a distended abdomen. In about 25% of cases a history of a sibling with the same syndrome is present. The stools are large, pale, foul smelling, and usually formed. X-ray of the lungs may show the chronic infection, while on examination of the duodenal juices a marked diminution or absence of trypsin will be found.

Anderson[5] divides the cases into three groups. In the first group, meconium ileus, inspissated meconium causes intestinal obstruction within the first few days of birth, producing symptoms of vomiting, abdominal distension and failure to pass meconium. It is believed that the inspissated meconium results from a failure of secretion of pancreatic enzymes into the duodenum. Early diagnosis is most important as Hiatt and Wilson[6] have shown that surgical treatment will save lives.

In the second group there is an early onset of respiratory symptoms, usually before six months of age, associated with the passage of abnormal stools.

In the third or 'late onset' group, respiratory symptoms develop after the age of six months and are also associated with symptoms of steatorrhoea. Children may not be brought for examination until the age of one or even two years. These are the cases that are mild and in whom the onset is insidious. Suddenly it is realized that the infant is not growing normally, that he is liable to frequent colds and coughs and that his stools are persistently abnormal.

May and Lowe[7] based their 'early' and 'late' groups on the age at which the infant is presented for treatment. Of 40 living patients 18 were brought for therapy after the age of one year. Until that time they had managed to get along comparatively well, and certainly without medical help. On careful questioning, however, it was discovered that both nutritional and respiratory symptoms had been present for a long time before. Hence in every infant with recurrent respiratory infection, steatorrhoea must be sought for and the stools observed repeatedly.

Aetiology. The aetiology and pathogenesis are not known. One theory is that the fibrosis of the pancreas is the primary lesion, and is congenital. It is always found in infants dying from meconium ileus.[8] The fibrosis leads to a deficiency of pancreatic enzyme production resulting in failure to digest food and thus causing steatorrhoea. It is suggested that this lack of enzyme, confirmed by the absence of trypsin in the duodenal contents, is caused by obstruction of the main pancreatic duct by an abnormal, viscid secretion. Stenosis or atresia of the pancreatic duct has been demonstrated[9] but it is uncommon. In two cases Baggenstoss[10] failed to confirm any obstruction in serial sections on the entire pancreas.

Persistently poor digestion of food, in the absence of essential enzymes, leads to malabsorption of vitamins and malnutrition. Vitamin A absorption is affected. This deficiency of vitamin A was discovered in this disease by Wilson and Du Bois[11], and Blackfan and Wolbach[12], Dafinee and others[13] considered it as the cause of the disease and suggested that the pancreatic pathology was secondary. Infants with this vitamin deficiency were found to have squamous metaplasia of the epithelium of the trachea and bronchi and it was suggested, therefore, that it was the cause of the respiratory complications. Very recently Anderson[14] has again discussed this important aspect of the disease and states that for the present, the evidence in favour of the nutritional hypothesis is indirect.

With the progressive study of this disease in the past 12 years it has become evident that other lesions occur, besides those in the pancreas and lungs. In some cases the secretion in the salivary glands has been found to be abnormal, while inspissated material has been discovered in the mucous glands of the trachea and bronchi, the small bowel and also the gall bladder. Local areas of cirrhosis have been described in the liver. In an attempt to correlate all these lesions in one pathology Farber[15] suggested an imbalance of parasympathetic or autonomic nervous control of pancreatic secretions and mucous glands. He was impressed by the widespread affection of mucous glands and so suggested 'Mucoviscidosis' as a name for the disease.[16] He considered the possibility of this being an enzyme deficiency disease, resulting in the production of an abnormal inspissating type of mucous.

More recently Baggenstoss and others[17] have postulated that the disease is caused by a congenital deficiency of secretin. They state that secretin is produced in the upper intestinal tract, stimulates the pancreas and so flushes and dilutes its secretions. In the absence of secretin the pancreatic secretions become thick, inspissated and thus cause blockage. The problem of whether this disease is due to malnutrition, or congenital maldevelopment of the pancreas, or enzyme deficiency, or some other congenital lesion, will only be solved by careful diagnosis and full investigation of numerous cases.

Diagnosis. This is essentially clinical and is confirmed by duodenal drainage. The duodenal juice lacks pancreatic enzymes especially trypsin. Sinclair[18], however, has pertinently recorded a case in which the duodenal juice was normal. Clinically the case was one of cystic fibrosis and on section of the pancreas only half of it was found to be fibrotic. This stresses that diagnosis must be clinical and must be considered along with coeliac disease, tuberculous mesenteric adenitis, lambliasis and severe chronic infection as mentioned by Sheldon[19]. Any infant who fails to thrive in the first few months, or presents with steatorrhoea or recurring respiratory infections may have a fibrosing pancreas. One must beware of the infants who are always 'chesty' and the others who have chronic bronchitis.

Prognosis and Treatment. The prognosis depends upon early diagnosis and is much better when treatment is started before the onset of respiratory infection. Since the introduction of penicillin, the prognosis has improved for those cases which have chronic lung infections, and May and Lowe[7] have suggested that it is the only important form of treatment and that dietary therapy is unnecessary. Very recently they have

reiterated this view[20] in an extensive and stimulating report. On the other hand Andersen[14] has shown that the early introduction of dietary therapy may prevent the onset of respiratory symptoms, and of 109 cases over a nine-year period, 72 are still living. For the present, therapy must consist of diet, pancreatin, antibiotics and full supplements of vitamins. Aureomycin may yet prove to be the best antibiotic to use.

CASE REPORT

The present case falls into the 'late onset' group, in that respiratory symptoms presented first at six months, but is unusual in that steatorrhoea occurred first at one year of age. The original diagnosis was asthma, then coeliac disease, then atypical pneumonia, and lastly cystic fibrosis of the pancreas.

S. V., a European female, was born at full term on 14 November 1945, weighing 7 lb. Although delivery was normal she would not suck well at first and was 'thin and miserable'. She was breast-fed and was given thyroid and later progressed well. There was one other female child of the marriage, aged 6½ years, who was perfectly well and there had been no miscarriages or stillbirths. There was nothing relevant in the family history.

At the age of five months, mixed feeding was introduced slowly without ill-effects. At six months she weighed 14 lb. 10 oz. and then she suddenly developed fever, cough and grunting respirations which responded well to sulphadiazine. At 6½ months she weighed 15 lb. 1 oz. She continued well until eight months of age when she developed a wheezy attack which was diagnosed as asthma and was controlled by ephedrine. These attacks, associated with rhonchi in the lungs, occurred on and off until the age of one year. She then weighed 21 lb., had four teeth, and was taking three meals per day.

It was at this time that the mother first noticed fatty stools 'like soup with blobs of fat on top' and later they became large, pale and evil-smelling. The infant became more irritable, had recurrent attacks of respiratory distress, failed to gain weight and periodically had 'loose stools'. These necessitated two admissions to hospital as 'gastro-enteritis', but at the age of 21 months she was diagnosed as a case of coeliac disease, and later spent three months in hospital. On discharge there was still some dyspnoea and it was at this stage that the writer was first asked to see the child, who was then two years four months old. She complained of a dry hacking cough, rapid respirations and pain in the right hypochondrium. Her appetite was good.

Clinical Examination. The general condition was fair and the respirations rapid. Weight 23 lb. Height 33½ inches. Head circumference, 19 inches. She was thin, but the buttocks were not wasted, and the abdomen was moderately protuberant. The liver was enlarged (3½ fingers below the costal margin). There were no adventitious sounds in the lungs. Nil else abnormal was detected. For the first time an X-ray of the lungs was taken and the report read as follows: 'There is fairly heavy clouding at the medial aspect of the right base extending up to the lung root, which is accentuated. The appearances suggest partial consolidation. The remaining lung fields show increased striation and a fine mottling. The appearances are not typical of tuberculous infiltration and the possibility that the condition is due to non-specific consolidation of a broncho-pneumonic type cannot be excluded.' She was admitted to hospital as primary atypical pneumonia, while tuberculosis and cystic fibrosis of the pancreas were considered in the differential diagnosis. The following investigations were done: White cells, 15,500 per c.mm. Polymorphs, 68%.; lymphocytes, 29%; eosinophils, 1%. Hb, 12.5%. Three stools were negative for ova and parasites. Urine, normal. Wassermann reaction, negative; Tuberculin Patch Test, negative. Intradermal Mantoux 1 : 1,000 and 1 : 100, negative. Sputa, negative for tubercle bacilli and fungi on six occasions.

Treatment consisted of a high protein, low fat diet, vitamin B complex and crude liver by injection, and a total of 25 gm. of sulphadiazine in five days. She improved and was discharged after 11 days but relapsed within six weeks. Re-X-ray showed no change in the lungs and she was readmitted as ? cystic fibrosis of the pancreas. Duodenal drainage was performed under fluoroscopic control and no trypsin could be detected in the duodenal juices.

The blood count was: White cells, 7,200 per c.mm.; Polymorphs, 65%, Lymphocytes, 28%; Monocytes, 6%; Eosinophils, 1%. Hb, 13%. Red cells, 4.7 millions per c.mm.

On this occasion treatment consisted of intramuscular penicillin plus caronamide, penicillin aerosol, sulphatriad, pancreatin gm. iii daily, vitamin B complex and crude liver extract by injection, vitamins A, D and C by mouth, and a high protein, low fat diet. There was a small gain in weight to 24 lb. 10 oz. The breathing improved and she was happier. For the first time crepitations were heard at the right base of the lung. The liver was still enlarged and the appetite was excellent. It was amazing to see the amount of food consumed with so little gain in weight.

At the age of two years nine months, there was another relapse with fever, a spasmodic pertussis-like cough, some cyanosis and adventitious sounds in the right lung. X-ray showed an extension of the lung pathology (Fig. 1) and so bronchoscopy followed by a bronchogram was performed by Dr. H. Grant-Whyte on 20 September 1948. There was much pus seen in the trachea and in both main bronchi, and as much as possible was aspirated. Lipiodol was introduced into the right main bronchus and produced a normal bronchogram. Culture of the pus was not helpful and on the assumption of penicillin-resistant organisms (four previous courses of penicillin had not been of much benefit) streptomycin gm. i daily was given for seven days and postural drainage instituted. The response was dramatic and for the next three months she was well. The cough had almost disappeared, the nights were peaceful, there were no fevers, the child was happy and there was a gain in weight of 2 lb. 8 oz. She continued with her treatment of all vitamins by mouth, high protein low fat diet, pancreatin daily and sulphatriad gm. ½ daily as a prophylactic. The stools

were of a normal colour and consistency but changed immediately extra fat was introduced into the diet.

In March 1949, at the age of three years four months, she had a mild upper respiratory infection with crepitations in both lungs. On 4 April 1949 she suddenly became extremely dyspnoeic, feverish and cyanotic and died within 12 hours of admission to hospital. The immediate cause of death was thought by the admitting officer to be either asphyxia or cor pulmonale.

An autopsy was performed and the bronchi of both lungs were found to be filled with pus. The pancreas did not feel particularly firm and appeared normal to the naked eye. The heart was normal and the liver showed some fatty change. The post-mortem diagnosis was acute bronchiolitis.

Section of the pancreas (Fig. 2) confirmed the diagnosis and showed marked fibrosis with destruction and dilatation of acini. A section of the lung was examined and reported upon as follows: 'There is definite hyperplasia of the mucous bronchial glands. The distension of the acini, the absence of mucus in the bronchi and the presence of few ducts proportionally, suggest that the mucin is not reaching the lumen of the bronchus. There is acute inflammatory change in the bronchioles extending into the lung substance and there is fibrosis in the parenchyma with cubical metaplasia of the alveolar lining in some parts of the tissue.'

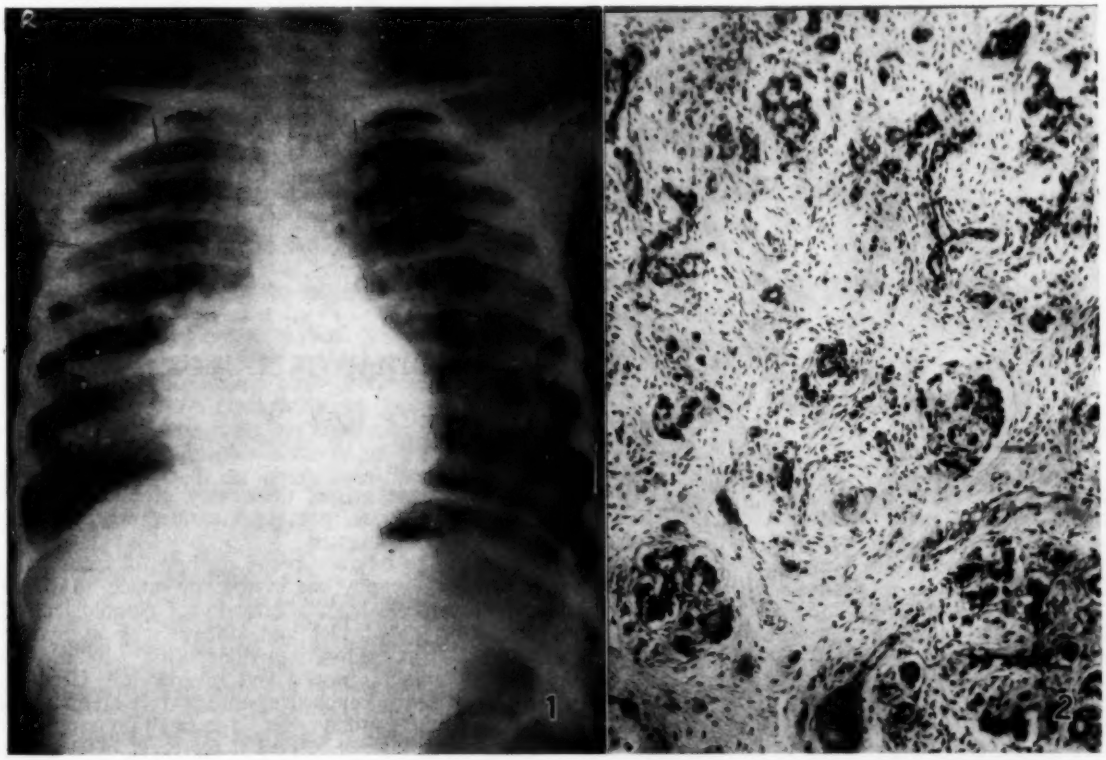

Fig. 1. P.A. view (11 September 1948). Note the opacity at the right base with striations running laterally and also towards the apex.
Fig. 2. Section of pancreas taken at autopsy. Note the whorls of fibrous tissue invading and distorting the acini and ducts. Some are dilated while others contain inspissated hyaline material.

SUMMARY

1. The theories of the aetiology of cystic fibrosis of the pancreas are reviewed.
2. The clinical diagnosis of the disease is stressed.
3. A plea is made for all children with recurrent lung infections to be considered as possibly suffering from fibrosis of the pancreas.
4. These children must be examined periodically.
5. An unusual case is reported belonging to the 'late onset' group and the pitfalls of the diagnosis mentioned.
6. Confirmation of the diagnosis is given in a section of the pancreas which shows the marked fibrosis that occurs.
7. The lung picture is that of any chronic infection and an X-ray plate is illustrated.
8. Treatment is discussed briefly and the importance of chemotherapy stressed.

ADDENDUM

Since completion of this report two further cases of fibrocystic disease have been diagnosed clinically. Duodenal intubation failed but the diagnosis was confirmed by the absence of trypsin in the freshly passed stools. This test, which has recently been described by Shwachman and others[21] is simple, easy to interpret and seems to be reliable.

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URETEROCELE SIMULATING BLADDER CALCULUS

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The diagnosis of a ureterocele, unless discovered accidentally by means of the cystoscope, depends upon how soon the complications to which it is prone call for systematic investigation by the urologist.

Ureteroceles may be congenital or acquired, unilateral or bilateral. The congenital cause is the most generally accepted because the incidence is greater in children and ureteroceles are so frequently accompanied by other congenital anomalies.

Inflammatory conditions may be responsible for ureteroceles. For example, Thompson and Greene report a patient seen by them in 1922 with granular cystitis and normal ureteric orifices, who had developed a ureterocele when cystoscoped thirteen years later. Freyer, in 1897, explained to the Royal Society of Medicine that calculi may be the cause of ureteroceles. According to the literature ureteroceles are seen most commonly between the ages of 9 and 16 years, but no age is exempt. O'Connor observed them in 2% of all cystoscopies, and Campbell in one out of 30 cases examined for pyelitis. Females and males suffer equally from the disease, which has been observed as frequently on the right side as on the left.

The pathogenesis is explained by the effect of a stenosed and obstructed ureteric orifice resulting in dilatation and atony of the lower end of the ureter, due to a congenital weakness of Waldeyer's sheath.

The inner layer of the ureterocele consists of ureteral mucous membrane, the outer layer of bladder mucosa, with an intermediate layer of scattered muscle fibres and connective tissue. The size varies from that of a small cystic swelling to a cyst filling almost the entire bladder.

They may produce no symptoms. On the other hand, by their very size they may interfere with the passage of urine through the internal meatus; or, when complications have arisen, these may produce symptoms such as frequency, pain in the side, urgency, incontinence and haematuria.

One has to rely on special methods of investigation, of which urography and cystoscopy suffice, to differentiate ureterocele from a diversity of pathological conditions of the bladder and kidney, such as hydronephrosis, pyonephrosis, pylonephritis, hydro-ureter, bladder tumours, stones and diverticula. Urography is by no means conclusive, and by this method alone one may miss the diagnosis. The small ureterocele that collapses during the period contraction of the ureter may be missed. The ureterocele may be continuous with a dilated ureter above, or it may be completely cut off from the vesical margin and thus not seen. It may appear as a filling defect in the bladder, the clarity of this depending upon the power of concentration of the kidneys. The same picture will be produced by the retrograde cystogram. Where the kidneys show good function, as in the case presented here, the ureterocele presents as a bulbous lower end of the ureter with a translucent halo surrounding it. On the other hand, when the cyst is of large size, almost filling the bladder, it may be merely an opaque halo surrounding the ballooned sac. Cystoscopy offers the most precise method of diagnosis if one bears in mind that tumours, polyps, bladder calculi and complications which have changed the appearance of the ureterocele may all cause confusion. Such a case was reported by Sauer, where the ureterocele was covered with triple phosphates, and only after tidal lavage with solution (G) was the appearance of the cyst clear enough to be recognized.

In its earlier stages the ureterocele presents as a cystic swelling with an appearance identical with that of the rest of the bladder mucosa, covered by similar

blood vessels. Its shape may be either round, longitudinal or flat, suspended by a broad pedicle overlying one or other ureteric orifice. In the earlier stages the size varies rhythmically, synchronizing with urinary effluxes. As years go by and as complications become superadded, oedema, fibrosis, calcareous deposits, ulceration and even gangrene may change the appearance to such an extent that diagnosis by cystoscopy becomes difficult.

Complications such as hydronephrosis, either unilateral or bilateral, infection, stone formation, and extrusion through the urethra, may develop at any stage of the disease.

Treatment. Should it be possible to introduce a catheter or bougie through the stenosed orifice of a small ureterocele, this is the method of choice. Cystoscopic scissors, cutting electrodes, fulguration, or a combination of any of these methods may be used, as in the case reported below. Fibrotic and rigid ureteroceles have been successfully treated by means of the resectoscope. Open operation (ureterocelectomy) was done in the old days before endoscopic instruments came into use. This may still be the best method in the case of large fibrotic and rigid ureteroceles or in the case of an extrusion through the urethra, or stone, where endoscopic instruments are used with difficulty. The radical operation (Nephro-ureterectomy) is reserved for advanced cases with secondary renal involvement.

CASE REPORT

On 5 October 1949 a male patient aged 51 years presented himself with complaints of dysuria, and a burning sensation at the end of the urethra towards the end of micturition. At the start of micturition the stream was strong and even, but towards the end became poor and liable to frequent interruptions, actually stopping at times and associated with a feeling of pressure over the bladder. A story of left-sided renal colic, two years ago, was volunteered by the patient, but he had no recollection of haematuria at any time.

General examination revealed nothing abnormal. The abdominal and rectal examinations were both negative. The urine was crystal clear with a Sp. Gr. of 1,012, and contained no sugar, albumin, blood or casts. Blood pressure, 130/90 mm. Hg; blood urea, 26 mg. per 100 c.c.

The most probable cause for the patient's symptoms was a bladder calculus. A straight X-ray was taken (Fig. 1) and this showed a stone lying in what appeared to be the lower end of the bladder. The size of the calculus created some doubt about its being a genuine bladder calculus because it was so small and an intravenous pyelogram was done before a litholapaxy was embarked on.

The intravenous pyelogram showed good function of both kidneys with normal pyelograms on both sides. The lower end of the left ureter presented a bulbous dilatation surrounded by a translucent halo (Fig. 2). The ureter above this was slightly dilated. The right ureter showed slight delay in emptying towards its lower extremity. After evacuation no stone was visible in the bladder and on partial emptying of the ureterocele the form of a calculus could be outlined in the cyst.

Cystoscopy (10 *October* 1949). Bladder capacity was normal. Overlying the left ureteric orifice, a large ureterocele was seen, covered mostly by bullous

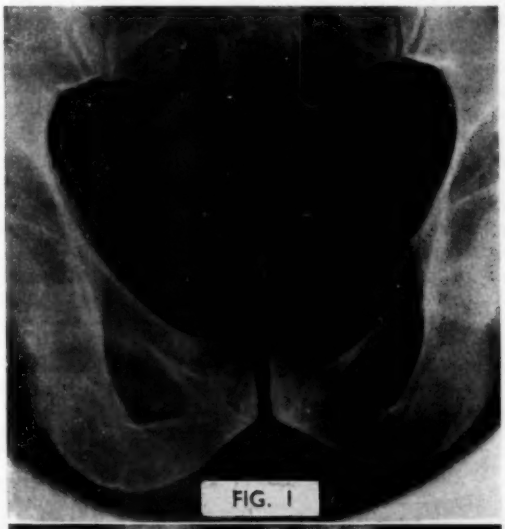

FIG. 1

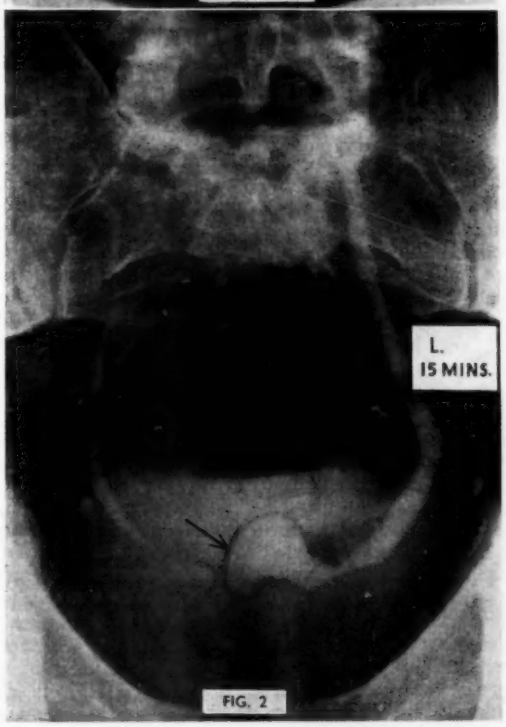

FIG. 2

oedema, especially towards the free end. On account of the bullous oedema it was not possible to make out the ureteric orifice. The right ureteric orifice was pinpoint in the presence of a minor degree ureterocele.

The rest of the bladder mucosa appeared normal and no stone was visible. The bladder neck, surrounded by bullous oedema, undoubtedly caused by irritation from the ureterocele, showed no other evidence of obstruction. The right ureteric orifice was dilated up sufficiently by meanst of ureteric catheters. Two-thirds of the length of the cyst wall was coagulated in linear fashion, finally opened towards the extremity by cutting current, and more extensive enlargement along the coagulated strip was done by means of cystoscopic scissors. Fibrosis and bullous oedema prevented complete collapse of the cyst wall.

An X-ray film of the bladder area taken seven days later showed a stone identical in size and shape with that previously seen in the ureterocele impacted in the prostatic urethra. On 7 November, under caudal anaesthesia, the stone was pushed back into the bladder and crushed. A panendoscope was used to examine the bladder before and after crushing of the stone and the débris washed away through the same panendoscope sheath. At this stage little was left of the ureterocele except a few remnants of its walls. The ureteric orifice was now satisfactory. It was later learned that progress was good.

Difficulties in diagnosis and subsequent treatment would have arisen unless one had adhered to routine investigation in this case, which simulated a bladder calculus so closely, both clinically and radiographically.

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CONTACT DERMATITIS DUE TO DDT

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In spite of its widespread use, reports of dermatitis due to contact with DDT are very few. Toxic effects on other organs are well known, however, and some of the symptoms and signs described include acute gastro-enteritis; coryza, cough and sore throat; giddiness, anxiety, apprehension, lassitude, muscular weakness; fibrillary, spastic and convulsive muscular contractions; aching pains; anaemia and white blood cell changes; increased blood calcium. In experimental animals there have been found degenerative changes in liver, spleen, kidneys and adrenals; gastritis and enteritis with petechial haemorrhages throughout the gastro-intestinal tract, and hyperplastic changes in the lymph follicles and Peyer's patches; pulmonary oedema and broncho-pneumonia; changes in blood vessels and cardiac muscle.[1, 2]

The risk of contact dermatitis from DDT in powder form is apparently slight. Schwartz, Tulipan and Peck,[3] writing in 1947, could find no reports of dermatitis as a result of exposure to DDT in factories where it was made, or in its use as an insecticide. Their investigations on workers employed in the manufacture of DDT seemed to indicate that it might be a weak primary irritant and sensitizer.

There have since been reported, however, a few cases of dermatitis due, apparently, to DDT in solvents[4, 5] (kerosene, xylene, dimethyl phthalate). Glassman and Buchan[6] state that although powdered DDT is non-irritating to the skin, solutions in bland solvents are absorbed by intact or abraded skin and can cause severe poisoning. They state that DDT is toxic but that the margin of safety is wide. The forms and conditions in which it is ordinarily used as an insecticide are not productive of toxic or dermatological effects unless grossly or accidentally misused. They remark further, however, that in common with many industrially used compounds, it can be surmised that the actual incidence of dermatological or systemic pathological changes is greater than recorded.

In France, DDT in powder form[7] and in solvents[8] has, since 1944, been used in considerable numbers of patients for the treatment of scabies. No toxic effects from these forms of treatment have been reported.

In a few recorded cases of dermatitis presumed to be due to DDT, the lesions produced have usually been of an erythemato-vesicular type, sometimes reaching a stage of exfoliative dermatitis. One case of periarteritis nodosa following exposure to DDT in kerosene has been reported.[9]

The interesting feature in the case now to be described is that the lesions were purpuric. Purpuric eruptions were sometimes seen in cases of 'dermatitis, contact, textile', during the war; and Hellier[10] mentions the possibility of DDT, impregnated in clothing, as a causative agent, but his investigations tend to demolish this theory.

Case History. The patient was a man aged 48 in good general condition and giving no history of any serious illness or skin disease before the onset of his present complaint.

Two years ago, about October 1947, he developed an itch of both legs and lower abdomen. Shortly afterwards there appeared a red eruption, of the same distribution, which became patchy, and in disappearing scaled a little and turned to a brownish colour. The whole attack lasted about one month. This attack, he believes, was related to the use of a sulphonamide powder as a local application after an operation on the left foot.

There was no recurrence until about October 1948,

when a similar attack occurred and cleared up in the same way in three weeks. On this occasion there was a transitory erythema of the face with marked oedema of the eyelids at the onset. This attack was preceded by a bout of gastro-enteritis for which he was treated with a sulphonamide by mouth.

The present attack began early in October 1949 with itching followed by the appearance of an erythematous rash on the legs, thighs, buttocks and lower abdomen, and later on the flexor surfaces of the arms, particularly about the elbows. No sulphonamides had been taken by mouth or applied externally since the last attack in 1948. When first seen, about a week after onset, the lesions of the lower extremities were closely set dull red irregular patches, up to an inch in diameter, dotted with small petechial haemorrhages. The legs and thighs were thickly covered by the eruption, but above this level the patches were more discrete and widely spaced. On the arms the lesions were few and small. At this time blood investigation showed 5,400,000 erythrocytes and 8,800 leucocytes (in normal proportions) per c.mm. The capillary fragility test was negative. A patch test with 5% sulphathiazole ointment gave a 2+ reaction after 24 hours.

The only treatment given in the next ten days, subjective symptoms being absent, was anthisan 0.1 gm. four times daily. The lesions began to fade towards a brownish colour and there appeared considerable superficial scaling especially about the knees. A mild keratolytic ointment was given but was only used twice as it provoked an acute erythema. At this time he noticed, when filling a spray apparatus with DDT 10% in kerosene, that his hands particularly and skin generally began to itch. Patch tests were made with the solution and with dry DDT powder. After 12 hours there was a 4+ reaction to the solution and a 3+ reaction to the dry powder. A patch test with another insecticide, benzene hexachloride in powder form, was negative.

The patient was advised to discontinue the use of DDT in his house and to have all old supplies removed. A month after the time of onset the lesions were fading gradually and the intervening skin of the lower extremities, where the rash was most pronounced, was a *café au lait* colour. Patch tests were repeated at this time. DDT powder 10% in talc gave no reaction; kerosene solvent and kerosene with 5% DDT both gave 1+ reactions after 24 hours.

A month later the original lesions had almost completely disappeared and no fresh lesions had appeared.

Discussion. It seems probable that the patient described was suffering from contact dermatitis due to DDT. As in similar cases previously reported DDT in a solvent was the causative agent. It is not certain whether all the attacks were caused by exposure to DDT, but it is interesting that the attacks began at the beginning of summer when insecticides are used in greatest quantity. It is possible that the gastro-enteritis which preceded the skin eruption in 1948 may have been a toxic effect of exposure to DDT.

DDT spray has been used in the patient's home as an insecticide during all the time in which he has been affected and it has been confirmed that spraying was begun and was most frequent in early summer. The most recent, and severest attack began while the patient was working, temporarily, in a hospital where the wards are regularly sprayed with a DDT solution and it can be accepted that he was exposed to this agent to a much greater degree than in previous years when he only encountered it in his home.

Sensitivity to a sulphonamide has also been demonstrated. This may be quite irrelevant, but it is possible that cross-sensitization has occurred, and this may have some bearing on the type of lesion produced as purpuric eruptions are not uncommon in sulphonamide-sensitive patients.

It is difficult in such a case to exclude the possibility that the solvent alone could be the causative agent; but the eruption was unlike anything usually attributed to contact with kerosene.

Summary. Contact dermatitis due to DDT is, considering the widespread use of this insecticide, very rare. The few cases so far reported have all been due to DDT in solution, the dry powder being apparently inactive as a skin sensitizing agent. An unusual feature in the case now reported is the presence of purpuric lesions.

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ASSOCIATION NEWS : VERENIGINGSNUUS

BORDER BRANCH

MINUTES OF THE ANNUAL MEETING OF THE BORDER BRANCH HELD AT THE WINDSOR HOTEL, EAST LONDON, ON SATURDAY 28 JANUARY 1950

Present: Drs. Johnston (*President*), Waddell (*President-Elect*), Wagner, Fleming, McConnachie, Geldenhuys, Segall, Pentz, Morgenthal, Schaffer, Norman, Navid, Lalor, Papilsky, Goldstone, Gutsche, Smyth, Alexander, Cunard, Meine, Haddad (*Acting Secretary*); *Visitor*: Dr. Sapirestein; *Apologies*: Drs. Hankins, van Schalkwyk, Gardiner.

Presidential Address: Dr. Johnston, in his presidential address, briefly outlined the outstanding events of the Association during the past year. He mentioned the new Cape Hospital Ordinance and the regrettable friction that occurred between the profession and the Medical Council over the Lipron Case. He suggested that more general practitioners on the Council would help to improve relations. Dr. Johnston also mentioned that a State Medical Service had not materialized and hoped it never would. He thought that Medical Aid Societies should be encouraged; 100% membership of the Association was very desirable for the good of the profession. He wished to thank the secretary, Dr. Hankins, for his work during the year.

Induction of New President: Dr. Johnston thereupon inducted Dr. Waddell as the new President for the ensuing year. Dr. Waddell thanked the meeting for the honour they bestowed upon him.

Election of Office Bearers: *President-Elect*: Dr. Schaffer. *Vice-President*: Dr. Norman. *Hon. Secretary and Treasurer*: Dr. Hankins. *Auditors*: McGillewie & King. *Branch Council Members*: Drs. Goldswain, Jaffit, Smyth, Nicolai, Segall, Marais, Berchowitz, Louw, Swemmer, Swift, Wagner.

Other Business: (*a*) A letter from Kingwilliamstown complaining that a certain Insurance Company was seeking a medical certificate without payment was considered. The Secretary was asked to send a copy to the Medical Secretary to hand to the Convener of the Contract Practice Committee for action.

(*b*) A circular from Medical Secretatry re Cape Hospitals Ordinance 1946 was read, giving information about how payments were made to hospitals in the Transvaal by the Provincial Administration. This was noted.

(*c*) A circular regarding insignia for Branch Presidents was read and noted.

(*d*) A circular regarding a General Practitioners Group was considered, and it was decided to refer the matter to the Divisions.

(*e*) A circular regarding the continuance of the existing Honorary System in the Cape Hospitals was considered. It was decided to refer this matter to the Divisions. It was further proposed by Dr. Segall and seconded by Dr. Schaffer, that: 'All circulars which are for the information of all members be printed in the *Journal*, reminding the Office that they promised all such articles would be put in the *Journal*.' This was passed unanimously.

(*f*) A letter from Medical Secretary asking for information regarding the charges made in the Divisions was read. The meeting decided to refer the matter to the respective Divisions.

Dr. Waddell wished to thank Dr. Ferguson for his work and write a letter of good wishes to him in his illness; also a similar letter to Dr. Hay-Michel.

A letter of condolence to be sent to Mrs. Bester, Port Elizabeth on the death of her husband.

The meeting adjourned in favour of the Annual Dinner which was enjoyed by all.

PASSING EVENTS

The Treacher Collins Prize Essay

Under the above title, the Council of the Ophthalmological Society of the United Kingdom has instituted a Prize of £100, awarded triennially, for the best Essay submited upon a subject selected by the Council.

The Prize shall be open to qualified Medical Practitioners of any nationality.

The Essay shall be written in the English language.

The subject for the next award of the Prize is:

Exophthalmos in Relation to Endocrine Disorder.

The closing date for sending in Essays for this award is 30 September 1951. Essays should be submitted to the Honorary Secretary, Ophthalmological Society of the United Kingdom, 45 Lincoln's Inn Fields, London, W.C. 2, from whom also any further particulars can be obtained. No name should be on any Essay, but a distinguishing pseudonym or quotation, which should be upon a sealed envelope containing the candidate's name and address, should accompany the Essay.

* * * * *

Mr. M. Cole Rous, F.R.C.S. was appointed an honorary consulting surgeon at the Groote Schuur Hospital, Observatory, C.P., in February 1950.

The other honorary consulting surgeons to the Hospital are Mr. T. Lindsay Sandes (appointed in 1946) and Prof. C. F. M. Saint (appointed in 1948).

International Congress on Diseases of the Chest

The first International Congress on Diseases of the Chest will be held in Rome from 17-20 September 1950. The Congress is sponsored by the American College of Chest Physicians through its Council on International Affairs, and is being held in the Carlo Forlanini Institute in Rome. The Italian government has pledged full support of the Congress, and physicians, chest surgeons and Members and Fellows of the American College of Chest Physicians are to foregather from all parts of the world.

Those interested in diseases of the chest are invited to attend and they should notify their wishes to Dr. D. P. Marais, Regent for South Africa, Southern Life Buildings, St. George's Street, Cape Town.

NEW PREPARATIONS AND APPLIANCES

Cyclobarbitone, A. & H.

Cyclobarbitone is 5-cyclohexenyl-5-ethyl barbituric acid. Though but slightly soluble in water, it dissolves easily in the alkaline fluid constituted by the mixture of secretions present in the intestines. It is therefore absorbed quickly through the intestinal mucous coat. Without acting so briefly as the 'basal narcotic', hexobarbitone, it is decomposed readily in the body and excreted more rapidly than barbitone or phenobarbitone, so that it acts for a shorter time than either of these two latter barbituric hypnotics and with decidedly less toxicity.

Clinical Effects. Cyclobarbitone induces moderately deep, moderately long sleep—lasting for 4 to 6 hours if the dose is 3 grains. It has no untoward effects on circulation or on respiration. During sleep some lowering of the respiratory rate occurs; and temperature falls but little. Cyclobarbitone does not produce unpleasant after-effects. The patient awakens rested and refreshed in mind and body. The drug is not cumulative in its action, since it is excreted so quickly. Apparently patients do not become habituated to it, and when it has to be given for a long time there is no need to increase its dosage.

Therapeutic Uses. Cyclobarbitone is of particular value in sleeplessness of mental origin, e.g. caused by worry or overtiredness (especially in elderly persons), by psychasthenia, or by hysteria. It has been found useful too in repeated loss of sleep through acute illness or toxaemia and in extreme and prolonged sleeplessness of physical origin, e.g. resistant insomnia caused by lesions of the brain, and insomnia of arteriosclerosis, of insanity, and after encephalitis lethargica. Given with an analgesic it quickly overcomes sleeplessness caused by pain. In rather large dosage it can do so alone if the pain is mild, though, like other derivatives of barbituric acid, it is not an analgesic.

Dosage and Administration. The usual dose of Cyclobarbitone is 3 grains. In mild insomnia, 1½ grains may be enough; and, in stubborn insomnia, 3 to 6 grains may be needed. The dose is best taken in a large cupful of warm or hot water or milk, in order to increase the rate at which the body absorbs it.

Cyclobarbitone Tablets, A. & H., gr. 3 are available in bottles of 25 and 100.

Further particulars are available from the manufacturers: Allen & Hanburys (Africa) Ltd., P.O. Box 860, Durban.

Dimorphid

'Dimorphid' (Dihydromorphinone) is a prompt and persistent suppressant of acute pain without the emetic and constipating side-effects of morphine. In normal doses there is no depression of mental faculties and, furthermore, 'Dimorphid' is less liable to cause vomiting or constipation.

Whenever morphine is indicated 'Dimorphid' can be used, especially for the relief of all types of severe pain, and to allay incessant cough without expectoration.

The oral dosage is half or one tablet increasing if necessary up to two tablets *pro dosi*. Parenterally it can either be given

subcutaneously in doses gr. 1/32 increasing in necessitous cases to gr. 1/16, or intravenously as an emergency measure where very acute pain is present.

'Dimorphid' is available in tubes of 10 oral tablets of gr. 1/25, in boxes of 4 × 1 cc. ampoules each of gr. 1/32, and in tubes of 20 hypodermic tablets each of gr. 1/20.

Made by Knoll Limited, London.

Sole distributors in Southern Africa: Scherag (Pty.) Ltd., P.O. Box 7539, Johannesburg.

AMPTELIKE AANKONDIGINGS

I. MEDIESE ASSISTENT-SEKRETARIS

Aansoeke deur tweetalige geneeshere word gevra vir die betrekking van Mediese Assistent-sekretaris van die Mediese Vereniging van Suid-Afrika by sy Hoofkantoor in Kaapstad.

Die salarisskaal is £1,000 × 40—£1,200 plus lewenskostetoelae teen Regeringskaal (£208 per jaar vir getroude persone en £57 4s. vir ongetroudes).

Van die suksevolle aansoeker sal verlang word dat hy by die Vereniging se pensioenskema aansluit.

Verdere inligting kan verkry word van die Mediese Sekretaris, Posbus 643, Kaapstad, aan wie aansoeke voor 30 Junie 1950 gerig moet word.

II. DEELTYDSE ASSISTENT-REDAKTEUR

Aansoeke deur tweetalige geregistreerde geneeshere word gevra vir die betrekking van deeltydse Assistent-redakteur.

Besoldiging (*nie-pensioendraend*): £100 per kwartaal plus 'n addisionele toelae van £15 per week wanneer hy gedurende die afwesigheid van die Redakteur waarneem.

Van die suksesvolle kandidaat sal verlang word dat hy minstens twee middae per week in die redakteurskantoor by Mediese Huis, Waalstraat 35, Kaapstad, aanwesig is. Van hom sal vereis word dat hy heeltemal vertroud raak met die redaksionele, kantoor- en advertensie-organisasie, by die kies en voorbereiding van manuskripte vir die drukker behulpsaam sal wees en dat hy by afwesigheid van die Redakteur met volle verantwoordelikheid optree.

Aansoeke moet gerig word aan Die Mediese Sekretaris, Posbus 643, Kaapstad, en moet hom teen 30 Junie 1950 bereik.

OFFICIAL ANNOUNCEMENTS

I. ASSISTANT MEDICAL SECRETARY

Applications are invited from bilingual medical practitioners for the post of Assistant Medical Secretary of the Medical Association of South Africa at its Head Office in Cape Town.

The salary scale is £1,000 × 40—£1,200 plus cost-of-living allowance at Government rates (£208 per annum for married persons and £57 4s. for single persons).

The successful applicant will be required to subscribe to the Association's Pension Scheme.

Further information may be obtained from the Medical Secretary, P.O. Box 643, Cape Town, to whom applications should be addressed before 30 June 1950.

II. PART-TIME ASSISTANT EDITOR

Applications are invited from bilingual registered medical practitioners for the post of part-time Assistant Editor.

Emoluments (*non-pensionable*): £100 per quarter plus an additional allowance of £15 per week when acting during the absence of the Editor.

The successful candidate will be required to spend at least two afternoons a week in the Editorial office at Medical House, 35 Wale Street, Cape Town. He will be required to become completely familiar with the editorial, office and advertising organization, assist in the selection and preparation of manuscripts for the printers and act with full responsibility in the absence of the Editor.

Applications should be addressed to the Medical Secretary, P.O. Box 643, Cape Town, and must reach him by 30 June 1950.

IN MEMORIAM

DR. M. FINE

The sudden tragic death of Mark Fine on Saturday afternoon, 4 March 1950, as a result of a motor accident, has deprived the profession in general and ophthalmology in particular of one of its most honourable members, in the prime of his life.

Born 47 years ago in Volksrust, he was brought up and educated there. He studied medicine and qualified at the London Hospital. After holding the usual appointments he became Ophthalmic House Surgeon and then proceeded to the resident appointments at the Royal Westminster Ophthalmic Hospital. He returned to South Africa early in 1927, and after a short period of practice in his speciality in Durban he settled in Johannesburg, where he remained in practice until his untimely death.

Of an unassuming disposition, he nevertheless more than pulled his weight, being ever ready to undertake more than the average amount of honorary hospital work. He became associated with the Ophthalmic Department of the Johannesburg General Hospital shortly after his arrival, in an unofficial junior capacity, there being at that time no official junior appointments. This did not deter him from giving of his best at all times, and his reward came only a few days before his death, when having climbed every step on the hospital staff, he was appointed one of the senior Ophthalmic Surgeons at the Hospital.

Mark Fine in his quiet way held very definite opinions of right and wrong, and all his colleagues who were privileged to be in his confidence can testify to his loyalty, fairness of mind and uprightness of character in personal and professional matters. He held strong and conservative views on matters of ophthalmological politics, and after many years of service as Secretary of the Ophthalmological Society of South Africa, he was elected President of the Society in 1948. It was the highest honour which his fellow ophthalmologists could bestow upon him, and he was very proud of the confidence thus shown in him.

In the practice of his speciality he was knowledgable and sound, and on his return from a trip to Britain and the Continent only a few months ago he was brimful of ideas for the improvement of the department in the hospital, which was always his main interest.

At the outbreak of World War II he was one of the first to offer his services, and he served as an ophthalmic specialist in the U.D.F. in the Union, Egypt, Tripoli and Italy. He was one of the first in and one of the last out, and during the periods of boredom in the Middle East one never heard a murmur of complaint, nor did his sense of humour ever fail him. To him 'things might have been much worse' was the motto—and his morale and good influence in the mess was recognized in his appointment as 'Entertainments Officer'.

He was unmarried, a devoted son and brother, and idolized by a large circle of friends. To his father, brothers, sisters and their families the sympathy of his colleagues goes out. He will be missed professionally and in the councils of our Societies.

REVIEWS OF BOOKS

THE PENICILLIA

A Manual of the Penicillia. By K. B. Raper and Charles Thom. (Pp. 875 + ix, with 172 figures and 10 colour plates. 91s. 6d.) London: Bailliére, Tindall & Cox. 1949.

Contents: 1. General Discussion. 2. The Manual Proper. 3. Reference Material.

This is a fascinating presentation of what is usually a very complicated, involved and rather dull subject. The differen-

tiation of individual fungi is made mainly on a morphological basis as in botanical classifications, in contrast to bacteriological classifications where the behaviour and the antigenic properties of these simpler organisms are the main points on which separation is made. The detailed study of morphological variation is somewhat tedious but in this manual has been made interesting by the introduction of a wealth of photographic plates. These show both the growth on culture media and the detailed structure seen by microscopy. The majority are bromide plates but there are also many in colour which are most vivid and beautiful.

The book begins with an historical outline of the study of Penicillia dating from Link's *Observationes* in 1809 to the present day. An enormous impetus to this study was provided by the demonstration of the potential therapeutic use of penicillin by Florey in 1940, following the important discovery of the antibiotic by Fleming in 1928. Because of the great importance of penicillin in medicine, the intensive study of the Penicillia, which had been limited to less than a dozen laboratories, was undertaken by hundreds of workers, mainly in America.

Then follows a very clear exposition of the descriptive terms used. This will prove of value to all workers in the mycological field, as will the detailed description of methods of cultivation, preparation of media, preservation and storage of cultures, etc. A chapter is devoted to a consideration of penicillin, the methods of its production, assay, chemistry and uses in medicine.

The main part of the manual is occupied by a detailed scheme of classification with keys to assist in diagnosis and useful summaries of the accumulated knowledge of the separate species. A comprehensive bibliography, conveniently arranged in sections, is attached.

The authors' aim was to produce a manual which would aid in identifying the Penicillia which may be encountered in the laboratory or which are significant in microbiological or biochemical processes. Their scheme of classification is based on a study of at least 4,000 cultures and the very considerable literature which has accumulated on the subject. The book provides interesting reading and the plates, particularly the colour plates, are really good. It is a most useful addition to the systematic study of the Fungi.

Better Hearing

Ways to Better Hearing—A Practical Guide for Anyone with Defective Ears. By Lowell Brentano, with an introduction by Arthur G. Wells, M.B., B.A., F.R.C.S. (Pp. 212. With illustrations. 10s. 6d.) London: George Allen & Unwin Ltd. 1948.

Contents: Part I. 1. 'Louder Please.' 2. True Confession. 3. Blueprint of the Ears. 4. Breakdowns and Bottlenecks. 5. Out of the World. 6. The Way Back. 7. Screen Test. 8. Machine-made Hearing. 9. Consumer Research. 10. Wear, Care, Repair and Beware! 11. The Junior League.
Part II. 12. Lip Reading and Exercises. 13. Educating your Hearing and Exercises. 14. How to Improve your Speech and Exercises. Appendices: 1. Speech Suggestions for the Hard of Hearing. 2. Psychological Aspects of Deafness. 3. Hearing Aid Dealers Approved. 4. Method of Rating the Performance of Hearing Aids. 5. Words and Music.

The author of this book is a layman, hard of hearing since childhood, following an attack of scarlet fever. It is written primarily for deaf patients, but is of absorbing interest to aural surgeons and anyone dealing with such patients.

While admitting that deafness is a severe 'cross' for anyone to bear, Mr. Brentano produces much sane and sensible counsel for the afflicted. He stresses that the condition should be diagnosed early by a competent aurist, and that the patient should accept his state without wasting valuable time in searching for useless remedies. When a hearing aid and lip reading are advised, the patient should, without delay, commence his rehabilitation, as the longer he remains at a low state of hearing acuity the more likely is he to develop handicapping psychological ills. What is more, the intelligent use of a hearing aid will keep alive the attention to sounds and the ability to derive meaning from them.

It is a strange fact that admission of diminished visual acuity is not considered a slur, and people will wear spectacles with good grace, but the mere suggestion to many deaf persons that they would be helped by a hearing aid brings an immediate negative response. The chapters in this book on the wearing and maintenance of a hearing aid contain an enormous amount of useful information and should do much to dispel the antipathy of the average deafened person towards these instruments.

Nearly half of the book is devoted to exercises in lip reading and in speech improvement, very helpful as an adjunct to instruction from a qualified teacher.

Ways to Better Hearing can be thoroughly recommended as a help to all deafened persons, to compensate for a good part of their loss, to acquire a realistic philosophy and to face the future with confidence.

Psychological Medicine

Psychological Medicine. By Desmond Curran, M.B., F.R.C.P., D.P.M., and the late Eric Guttman, M.D., M.R.C.P. (Pp. 252 + viii. With illustrations. 12s. 6d.) Edinburgh: E. & S. Livingstone Ltd. Third Edition.

Contents: 1. Introductory. 2. Actiology of Mental Disorders. 3. Symptoms in Mental Disease. 4. Psychiatric Case-Taking. 5. Treatment. 6. Constitutional Anomalies. 7. Organic Syndromes. 8. Psychiatric Aspects of Head Injury. 9. Drug Addictions. 10. Schizophrenia. 11. The Effective Reaction Types. 12. Obsessional States. 13. Hysterical Reactions. 14. The Legal Aspects of Mental Illness.

This is the third edition of a well-written, easy-to-read concise but comprehensive 'textbook' of psychological medicine or psychiatry, which the authors define as: 'That branch of medicine whose special province is the study, prevention and treatment of all types and degrees of mental ill-health, however produced'.

The sound approach to their subject is at once deduced by this early passage: 'A thorough physical examination is rightly regarded as essential; but a reasonably thorough assessment of the mental and emotional aspects of all patients should be regarded equally essential; and it can usually be achieved without undue difficulty by taking a careful history with the requisite background of knowledge'. The authors rightly condemn the over-elaborate, repeated 'investigations' of patients; for the psychiatric diagnosis should be made as much on the positive assessment of the patient by a competent clinician as on the negative reports produced by laboratory technicians.

One of their explanations for the undue delay in the recognition of psychiatric illness is that physicians 'are confronted with the predicament that to make a psychiatric diagnosis leaves them with no very clear idea of what can or should be done'. They point out that the mass of minor psychiatric ailments can be treated by general practitioners and that most patients do not need prolonged analysis. Also, that many patients who are not curable are eminently treatable.

Essentially, the book shows what kind of approach is necessary to psychiatry. 'The question to be asked about a psychiatric patient is not, therefore, "What kind of mental disease has this patient got?" but rather, "What type of reaction does this *individual* show?"'

'Disease entities' are replaced by 'reaction types' or 'psychiatric syndromes'. Nevertheless, the authors are too experienced and commonsensical to fall into the 'doctrinal' trap of the rigid Freudian system of psycho-analysis, which has also become 'a school of philosophy'.

The most important part of psycho-therapy, they write, is dependent upon the ability to adapt explanation not only to the patients intellectual level, *but also to his emotional needs.* This cannot be over-emphasized.

This is a book which can with advantage be read, together with other works recommended in the excellent bibliography.

Students will like it for the neat way in which mental illnesses are classified and docketed without fuss as well as without favour to various 'schools of psychology'. The medical tradition of this book is essentially British.

DIGESTIVE DISEASES

Handbook of Digestive Diseases. By John L. Kantor, M.D., F.A.C.P., and Anthony M. Kasich, M.D., F.A.C.P. Pp. 658. With 149 illustrations. $11.) St. Louis: The C. V. Mosby Company. 2nd ed. 1949.

Contents: 1. Classification of Digestive Disorders. 2. Diagnostic Methods: History and Physical Examination. 3. Diagnostic Methods: Special Tests. 4. Organic Constitutional Inferiority: The Anomalies. 5. Diseases of the Mouth. 6. Diseases of the Esophagus. 7. Diseases of the Stomach. 8. Diseases of the Stomach (continued). 9. Diseases of the Stomach (continued). 10. Diseases of the Stomach (continued). 11. Diseases of the Small Intestine. 12 Diseases of the Small Intestine (continued). 13. Diseases of the Colon. 14. Diseases of the Colon (continued). 15. Diseases of the Appendix. 16. Diseases of the Rectum and Anus. 17. Diseases of the Liver. 18. Diseases of the Liver (continued). 19. Diseases of the Liver (contineud). 20. Diseases of the Gall-bladder and Bile Ducts. 21. Diseases of the Pancreas. 22. Intestinal Parasites. The Protosoa. 23. Intestinal Parasites (continued). The Flatworms: Cestodes. 24. Intestinal Parasites (continued). The Flatworms: Trematodes. 25. Intestinal Parasites (continued). The Roundworms. 26. Digestive Symptoms in Extradigestive Diseases. 27. Psychiatric Aspects of Digestive Diseases. 28. Gastrointestinal Allergy.

This a new edition of Kantor's original *Synopsis of Digestive Diseases.* The *Synopsis* was published about 10 years ago and proved to be a new approach in the handling of gastro-intestinal diseases. It was straight-forward, concise and methodical and, being a synopsis, it was a small book. It was, however, much more than a lecture notebook for concise teaching. It was an original book written by a great authority on his subject and it contained much of the author's own work on certain aspects of gastro-enterology. It was not designed to replace the larger textbooks on the subject and as a concise account of gastro-intestinal diseases it was of great value.

When Kantor died the task of completing the second edition fell to Dr. Kasich. Now the book is presented in a somewhat different form. It is much larger, but the new author has maintained the general principles of Kantor's approach. There is a systematic description of gastro-intestinal diseases with a sound physiological approach to the problems frequently encountered in these conditions. The book has also the great merit of describing the latest research in many fields. Many of Kantor's original, simple diagrams are reproduced and the sections on irritable colon and constipation (on which subjects Kantor contributed so much) are reproduced from the old work. The book has been enriched by the reproduction of many new photographs, drawings and X-ray plates and has attached a valuable bibliography.

Although the replacement of Kantor's original book is regretted, it was inevitable that a second edition would have to be expanded to include much new knowledge and more detailed description.

This book is now full enough to be used as a textbook. It is, however, still concise and extremely readable and can be warmly recommended as a good, all-round work on the subject. It should be very widely appreciated.

SIGHT, LIGHT AND EFFICIENCY

Sight, Light and Efficiency. By H. C. Weston, F.I.E.S. (Pp. 308 + xiv. With 132 illustrations including 13 coloured plates. 42s.) London: H. K. Lewis & Company, Limited. 1949.

Contents: 1. The Sense of Sight. 2. The Causes and Symptoms of Eyestrain. 3. Occupational Demands upon Sight. 4. The Facilitation of Visual Tasks. 5. Lighting and Visual Efficiency. 6. Incentive Luminance and Colour. 7. Testing Sight for Work. 8. The Protection of Sight at Work.

Light and shade, colour and contrast, in ever-varying pattern, make up our visual world. We have learned to lighten our labours in numerous ways, but we are only now learning to appreciate the comfort of adequate light and to exploit the amazing efficiency of our hardest-worked sensory organs—the eyes.

This is a book about eye-sight and the demand made upon it by industry and occupation, by one who is both an expert in occupational optics and an illuminating engineer. He has carried out an investigation for the Medical Research Council at the Institute of Ophthalmology and his findings are set out here in most orderly fashion. He tells how the influence of colour and contrast, of shadow and light, of movement and of distance will decide whether a task is exacting or not, without going into a mathematical maze. Everywhere there are excellent photographs and illustrations to show how the eyes are ill-treated in certain occupations, how proper illumination may change a task from drudgery to pleasant toleration, at least. The mainstay of the industrial illuminator to-day is the now suddenly ubiquitous tubular fluorescent lighting. Its spectral quality approaches that of day-light and its low brightness factor compared with its illuminating power renders it so far the most glareless type of lighting, although it has obvious defects. The maximum use of natural day-light, where possible, is emphasized.

The book begins with a sketchy anatomy and physiology of the eye as well as a dissertation on eye-strain and optics, all of which seem rather unnecessary. These chapters have been revised by Sir Stewart Duke-Elder himself. One reads with surprise that he has passed the term posterior chamber for what is obviously intended to be posterior segment or vitreous cavity.

The last chapter contains an excellent review of industrial eye injuries and their prevention. The book is eminently readable and is itself a good example of a comfortable visual task. It will meet with the hearty approval of every reader in the comfortable use and maximum efficiency of the eye at work—and the reader will be in good company, because it has the patriarchal blessing of Sir John Herbert Parsons himself.

OCCUPATIONAL OPHTHALMOLOGY

Occupational Eye Diseases and Injuries. By Joseph Minton, F.R.C.S. (Eng.). (Pp. 184+viii, with 24 illustrations. 21s.) London: William Heinemann Medical Books Limited. 1949.

Contents: 1. The Nature, Growth and Extent of the Problem. 2. The Incidence of Eye Injuries in the Metal and Engineering Trades. 3. Types of Eye Injuries. 4. Contusions of the Eye, Delayed Optic Atrophies and Fractures of the Orbit. 5. Chemical Eye Injuries. 6. Acute and Chronic Occupational Keratitis and Conjunctivitis. 7. Toxic Effects from Industrial Solvents and other Industrial Poisons. 8. Radiant Energy and the Effects on the Eye. 9. Miners' Nystagmus and Eye Injuries at Coal Mines. 10. Vision and the Selection of Personnel in Industry. 11. The One-Eyed Worker. 12. The Employment of the Blind and Partially Blind. 13. Illumination in Industry. 14. Colour in Industry. 15. Eye Protection. 16. Prevention of Eye Injuries.

The main impression on reading this excellent little book is twofold: firstly, the variety of industrial and occupational afflictions of the eye; secondly, the relative infrequency of serious eye injuries when one considers the many hazards to which the eyes are exposed. This is a tribute to accident-preventing devices, now insisted on in most occupations.

Chemical injuries of the eye are, perhaps, too sketchily dealt with, but the interesting effects of radiant energy on the various eye structures are fully treated. The increasing use of organic solvents in industry, usually of a volatile nature, has led to an increase in optic nerve damage. One is pleased to note that the author is prepared to defy tradition and condemn the use of atropine in transient corneal injuries which heal up far sooner than the uncomfortable effects of the mydriatic wear off.

Not the least interesting part of the book deals with the employment of one-eyed, semi-blind and blind persons in industry. It has been shown conclusively that, with certain reservations, the one-eyed man is in no way handicapped in industry, or more accident-prone than his two-eyed brother; and yet he is often forced into a lower earning capacity on account of ignorance and prejudice. For the partially and totally blind there is a surprising number of occupations at which they are competent and happy.

The author makes no effort to discuss pathology and treatment. First Aid only is indicated here. The book will be a useful addition to any industrial doctor's library.

TUBERCULOSIS NURSING

Tuberculosis Nursing. By Jessie G. Eyre, S.R.N., B.T.A. (Hon.). (Pp. 291 + xii, with 103 illustrations. 21s.) London: H. K. Lewis & Co. Ltd. 1949.

Contents: 1. What is Tuberculosis. 2. The Course and Clinical Picture of Pulmonary Tuberculosis. 3. Sensitivity and Immunity. 4. Symptoms and Diagnosis of Pulmonary Tuberculosis. 5. General Principles of Treatment of Pulmonary Tuberculosis. 6. The Hygiene of the Ward. 7. General Nursing Procedures. 8. The Feeding of the Tuberculous Patient. 9. Special Nursing Procedures. 10. Chemotherapy in Association with General Treatment. 11. The Administration of Medicines and Drugs. 12. Collapse Therapy. 13. Collapse Therapy (continued) and Chest Surgery. 14. The Nursing and Treatment of Pleurisy and Empyema. 15. The Sequelae of Pulmonary Tuberculosis. 16. Tuberculosis of the Bones and Joints. 17. The Dying Patient. 18. Tuberculosis Care and After-care Schemes. 19. Appendices.

This is an excellent volume from the pen of an experienced sister tutor. Teaching nurses has led her to produce a monograph notable for its orderly classification and its clarity and simplicity of style. The section on tuberculosis of bones and joints, with the appendices, indicates much practical experience on the part of the writer, who supplies many useful hints on the orthopaedic problems involved. The problem of tuberculosis and its management is discussed in the light of up-to-date knowledge of the latest advances in treatment, and without revealing any bias in favour of a particular method.

This little volume should be in the hands of every nurse who desires to be proficient in her work.

RURAL EPIDEMIOLOGY

Epidemiology in Country Practice. By W. N. Pickles, M.D., (Lond.). (Pp. 112. 10s. 6d.) Bristol: John Wright & Sons Ltd. Re-issued 1949.

Contents: 1. An Appeal. 2. The Lines of Communication. 3. Technique. 4. The Commoner Diseases. 5. Chicken-Pox and Shingles. 6. Some Dysentery and the Rarer Diseases. 7. Epidemic Catarrhal Jaundice. 8. Epidemic Myalgia.

This little volume is a reprint, unaltered, of the edition first published some 11 years ago when it became a standard account of rural epidemiology. The re-issue was necessary because the entire stock and the type of the book were destroyed by enemy action in the early part of 1941.

The publisher's decision to bring out this volume again is, indeed, a wise one. Dr. Pickles has demonstrated not only the great contribution to the understanding and the control of the common communicable diseases which the general practitioner can make, but he has also illustrated in his theme the fact that there is considerable room for sound clinical research and observation which only the general practitioner is in a position to contribute to the general body of medical knowledge.

The book is beautifully produced and will delight any practising doctor by the charm of its style as well as the attractiveness of the demonstration of the principles of common sense in tackling the ills around us.

CORRESPONDENCE

SECOND SEMINAR ON WORLD HEALTH ORGANIZED BY WORLD FEDERATION OF UNITED NATIONS ASSOCIATIONS

To the Editor: You will recall that during the Second World Health Assembly last year at Rome, a 'Seminar on World Health' was organized by the World Federation of United Nations Associations. Its purpose was to acquaint a number of selected medical students and young physicians with the activities of WHO, and, generally, with international health problems. There were 25 participants from 10 countries. Medical and public-health specialists who had come to Rome to attend the Health Assembly lectured and conducted discussion groups. The participants were also given the opportunity to attend sessions of the Health Assembly and of its main committees.

Since this first 'Seminar on World Health' was generally regarded as highly successful, the WFUNA has decided to plan a similar gathering this year which would take place at Geneva from 8-19 May, i.e. during a part of the forthcoming Third World Health Assembly. Following the pattern set in 1949, participants will be selected through national and student associations affiliated with the WFUNA in the various countries.

The WFUNA will be responsible for all arrangements, but is not in a position to give any financial assistance to participants. It has therefore been suggested that, as was done last year in several instances, governments may wish to provide grants-in-aid for individual medical students and young physicians who desire to attend the 'Seminar' but would otherwise be unable to do so.

It should be noted that the World Health Organization will not be associated officially with this undertaking. However, the significance of such gatherings as a means of developing an informed opinion on WHO among the rising generation of the medical profession is considered to warrant the Organization's full co-operation and moral support.

All correspondence regarding the 'Seminar on World Health' should be addressed to:

World Federation of UN Associations,
Education Commission,
UNESCO House,
19 Avenue Kléber,
PARIS 16e,
France.

World Health Organization
Palais des Nations
Geneva
23 February 1950

Brock Chisholm, M.D.
Director-General

X-RAY TREATMENT IN DERMATOLOGY

To the Editor: I would like to answer, from the dermatological angle, Dr. Charlton's recent paper on the X-ray treatment of non-malignant diseases, published by you on 4 February. I do not wish to appear unduly critical, but in the science of dermatology, possibly even more than in any other branch of medicine, there may be more than one form of treatment for certain conditions, and it is impossible to say that one is always right or another always wrong, and in view of the fact that some of the dosages mentioned in Dr. Charlton's paper might possibly be not without danger in less experienced hands than his, I feel it would only be fair to present the generally accepted views held by modern dermatologists, as opposed to those held by some radiologists. I will classify them under separate headings, as Dr. Charlton has done.

Haemangiomata: Unfortunately, in his article Dr. Charlton does not classify these, but merely mentions that treatment with superficial X-rays with a low K.V. is effective. This I know is so with the superficial cavernous haemangiomata, but it is also recognized now by the vast majority of dermatologists that these lesions—so-called strawberry naevi—practically always disappear spontaneously, leaving very little scarring. I admit that a skilled radiologist can achieve a good result cosmetically, without the anxiety of waiting, but to err is human for us all, and although to forgive may be divine, the skin seldom does so, and there must be few dermatologists of any experience who have not seen some tragic scarring from such treatment. When Nature can do the job very much more safely than we can, it seems a pity not to leave it to her, and that is, I think, the view adopted by most experienced dermatologists to-day.

With regard to the capillary type, the so-called port-wine stains, the general opinion among dermatologists is that radiotherapy, to be effective to any degree, will have to be given in such a dosage as to leave unsightly atrophy and telangiectasia. McKie and Cipollaro, usually the staunchest of protagonists for superficial radiotherapy in dermatology, are even opposed to Grentz ray and Thorium X treatment for

capillary haemangiomata, although the latter, especially Thorium X, is advocated by many dermatologists for at least producing some cosmetic improvement.

Warts: I would say that about 70% of plantar warts can be cured by most of the accepted conventional treatments, and I think that few radiologists or dermatologists will claim, at best, more than 80-85% cure with X-rays on a careful follow-up. Certainly I myself, in common with other dermatologists, have seen a large number of plantar warts still present in spite of heavy X-ray treatment. Dr. Charlton recommends treatment spread over three weeks, using a low K.V. Chaoul technique, with a total dosage of 2,250 r to 3,600 r. I would suggest that this is a dangerously high dosage, and that if one is reduced to X-ray treatment, if there is no result from one treatment with, say, 600-900 r with the same K.V., to be repeated if necessary in a month's time, then it is wisest to consider an alternative line of therapy. Any further radiological treatment merely increases the risk of a third-degree X-ray burn. These latter, I am afraid, are by no means as rare as they ought to be, and in a comparatively short period of dermatological practice in this country, I have unfortunately seen four such cases, which present a very difficult problem for treatment.

As Sir Archibald MacIndoe pointed out in a recent paper read to the Royal Society of Medicine, the chronic X-ray ulcer is all too common. In his series he cited over 1,000 such cases seen by him. The majority had originally been treated for warts, an innocent neoplasm.

His experience was that patients who developed such lesions seldom returned to the radiologist or dermatologist who treated them, but tended to turn rather to other dermatologists or plastic surgeons. When he read this paper numerous radiologists present protested that they had had no such ill-effects as a result of treatment, but he was in several instances able to name patients in his series who had been treated by the radiologists concerned, and developed this complication. I would suggest that X-rays be reserved for only the more intractable plantar warts of the non-mosaic type, and then given in a rather more conservative dosage, as suggested above.

For the ordinary common wart elsewhere on the body, I would suggest there are many treatments as effective as, and a good deal safer than X-ray treatment. The chief appeal of X-rays to the patient is, of course, the absence of the immediate pain of any operative procedure. The risk of subsequent late sequelae is too often ignored or not appreciated.

Hyperkeratoses: Unfortunately, Dr. Charlton has not described what he means by these, but I imagine he is referring to the actinic or senile keratoses which show a tendency to squamous-celled malignant degeneration. I would like, however, to point out that the X-rayed keratoses which are seen after third degree reactions are very similar histologically to the above, and also show the same tendency to malignant degeneration. In each case the histopathological picture is very similar. As a result of this there has been a general trend among dermatologists recently to avoid the use of X-rays for treating keratoses, and to remove them with diathermy or electro-cautery.

Moles: I cannot agree with Dr. Charlton that the most satisfactory treatment for moles is diathermy coagulation. It is generally accepted that the ideal treatment for moles is, if possible, masterly inactivity, but if for some reason they are required to be removed, wide and deep surgical excision is indicated, if necessary with skin grafting. I myself have seen three cases of rapid fatal dissemination following interference with a mole which gave no indication of being malignant at the time of operation. In two cases CO_2 snow had been used, and in the other, electro-coagulation. Other more experienced dermatologists have seen more cases, and such tragedies as these should, I think, be a deterrent from any interference with a mole. Certain types of malignant melanomata have, however, been said to respond to radiation; but on the whole I would say that radiotherapy has little place in the treatment of moles, benign or malignant. In the malignant melanomata, wide excision locally with subsequent block dissection of the regional lymph glands would, I suggest, be the method of choice.

Leukoplakia: Here I would suggest dividing the cases into two groups. The smaller superficial lesions of leukoplakia are best left untreated beyond removing any causes of local irritation of the mucous membrane. They seldom become malignant, and tend anyway to recur after X-ray therapy. The larger hyperkeratotic type, which tend to be pre-cancerous, are treated by many radiologists as though they are malignant lesions, and the routine used by Dr. Sacks at the Addington Hospital is that of 800 r with a comparatively high K.V. given daily for six to eight days. It is, of course, most important, when making the diagnosis of leukoplakia, not to confuse lichen planus of the mouth with this condition. There should be no such confusion, but I have seen cases of lichen planus referred to radiologists for 'treatment of leukoplakia'.

Pruritus Ani and Vulvae: This relatively common dermatological problem may be due to a great variety of causes, and I would describe it as a symptom rather than as a disease. It therefore should never be treated without treating the underlying cause at the same time. If we eliminate causes due to some obvious pathology, such as fungus infection, diabetes, worms, trichomonas infection, seborrhoeic dermatitis and other causes, we come down to probably the commonest single cause, which is a frank neurodermatitis. Dr. Charlton recommends X-ray treatment without, apparently, any other approach, but I, in common with other dermatologists, have unfortunately seen only too many patients with this complaint who have already received as much X-ray treatment as the prudent dermatologist considers advisable, with either little, or only transient, improvement which has probably lasted for a few weeks, or at the most, months. Our task then is immeasurably harder. The patient is disillusioned and depressed, and, furthermore, if we overcome the increased difficulties and produce considerable improvement by conservative treatment, there is often a stage at which they appear to stagnate, and it is at this stage that small doses of X-rays to help them over the stile would be invaluable. This treatment, however, may be contra-indicated owing to the previous treatment, and even if not contra-indicated, the response to a second irradiation is not as satisfactory as on the first occasion.

I would suggest that pruritus ani or vulvae should always be investigated by a dermatologist first, and only receive X-ray treatment on his advice. In these cases, to concentrate one's attention on the itching skin is to miss the whole significance of the syndrome. The pruritus is merely a symptom of something deeper within, and to cure the condition it is much more important to attack the latter than the former. If this is done, and some suitable anti-pruritic topical application given with sedatives, many will clear up without any further treatment, beyond explaining the cause and the mechanism of the trouble, and other such simple psychotherapy.

Alopoecia areata: This condition is not so rare as Dr. Charlton declares, being seen quite frequently by dermatologists, and of course the vast majority clear up spontaneously, as it is most frequently associated with some temporary emotional disturbance. Thorium X has been used with some quite good effects, but owing to the disease's tendency to spontaneous cure it is difficult to assess the value of this treatment. I am rather surprised that Dr. Charlton recommends such hard rays in his treatment, and I think most dermatologists would be chary of using this method. Errors in dosage are never impossible, and in such a case carry a very heavy penalty.

Acne: I note that a course of 1,200 r over a month is recommended, to be repeated after three months. Here again, relatively hard rays are advised, and I think many dermatologists and some radiologists would consider this dosage rather dangerously high, with all too frequently the improvement being only transient. Radiotherapy in acne is much too debatable a subject to be gone into in such a short space, but dermatologists of repute are by no means unanimous in their appreciation of its permanent benefit.

Fungus: I think it is generally agreed by all dermatologists that X-rays have no fungicidal effect. The error has probably crept in from their use in tinea tonsurans, where they effect a cure solely by their capacity to produce epilation, and fungicides are required after this. With regard to alleged improvement in fungus infection of the hands, I think many of these are probably 'ide' eruptions secondary to fungus infection elsewhere, and one would naturally expect this to improve with X-ray treatment, although it is not the method of choice

until the sensitizing lesion has been dealt with, after which anyway the cheiropompholyx would usually clear up on its own. The prompt improvement on the feet in certain cases may be due to areas of localized eczematous sensitization to a mycotic infection of the skin having been mistaken for a widespread mycotic infection. In such cases frequently the sensitization area will clear up while the infected area remains unaltered.

I am far from wishing to cross swords with our radiological colleagues; rather I would suggest we can help each other in the superficial X-ray treatment of skins. Each has specialized knowledge which should be pooled, and each can learn from the other's experience.

It is rather interesting to note that in recent years in several of the London teaching hospitals the superficial X-ray therapy for diseases of the skin (other than malignancy) is ordered by the Dermatological Department, and the dosage controlled entirely by them. This lead has been followed, I believe, in Copenhagen and elsewhere on the Continent. It is also rather significant to note that, with a few exceptions, the more experienced dermatologists are becoming more and more cautious with their radiotherapy, and if asked for their reasons they admit that they have seen so many late ill-effects of incautious radiotherapy that they themselves veer more and more in the other direction. I think, however, most dermatologists will admit that all malignant conditions of the skin are better treated by a radiologist with his highly specialized knowledge and experience.

I would, however, put forward a plea that radiologists and dermatologists in general should work rather more closely together than we do at present in this country. I would suggest from my own personal experience and that of other dermatologists of far wider experience, that the dermatologist is usually better able to assess the ill-effects of radiotherapy than the radiologist. Unless a most rigorous follow-up system is employed by the latter, the vast majority of patients who have received no benefit from X-ray treatment of their skin tend to go elsewhere without informing the radiologist. Few of the failures seen by dermatologists have returned to the radiologist. On the other hand, quite naturally, the successfully treated patients return enthusiastically to record their satisfaction at the relief of their symptoms. If, at a later date, they relapse again, they also tend to go elsewhere, especially if they have been advised that they have received the maximum dosage considered safe for that area of skin, and in this connection I would like to suggest to Dr. Charlton that the smaller doses, such as 75 r to 100 r given weekly to such conditions as chronic prurigo, are, in the opinion of most dermatologists, just as effective as larger doses, and if no improvement is achieved with these small doses, larger doses very rarely help.

Finally, I would like to record one incident that occurred at Guy's Hospital when a new superficial X-ray plant was installed. Owing to some error (only discovered four weeks after the plant had been in use), a thick filter entirely excluded all rays. When this was discovered an immediate check-up was made of all the patients treated, and we were rather interested to find that over 50% of the patients treated, when interviewed, stated that they had derived a very great benefit from the treatment! We must not forget the very high functional element in a large number of chronic skin diseases.

John Leeming, M.D. (Oxon.).
415, Colonial Mutual Buildings,
330, West Street,
Durban.
1 March 1950.

PENICILLIN AND 'BLACK TONGUE'

To the Editor: Recently in the press I noticed a paragraph on 'Black Tongue'. It is alleged that this condition has had doctors puzzled for a long while. Now it has been found to be due to penicillin taken orally.

I had a patient with a sore throat and he asked me to let him have some penicillin lozenges, which I did. A short while after he had used it he developed a thick black fur on the tongue, extending from the taste buds, which appeared to be enlarged, almost to the tip. The condition was most marked down the midline of the tongue. It persisted for nearly five weeks. It slowly wore off at the edges, but the most dense part near the posterior part of the tongue stripped off in one sheet. In parts the 'hairs' were almost a quarter of an inch long, and they hindered the act of swallowing.

About two months later he once more used penicillin lozenges. Again the coated tongue developed while using the tablets. This time, though, it was white and almost as dense and long as the 'black tongue'. After stopping the penicillin and taking large doses of vitamin C, the 'fur' started coming off in bits and pieces, some being about 5 mm. in diameter. The 'black tongue' stripped off after using vitamin C and riboflavin for five days. The piece that came away was about the size of a florin.

R. Momberg.
United Buildings,
Windhoek, S.W.A.
10 March 1950.

THE LATE DR. MARK FINE

To the Editor: Those of us in England who knew 'Markie' Fine when a student and house surgeon at the London Hospital, and as house surgeon and registrar at the Royal Westminster Ophthalmic Hospital, and who met him during his service in Egypt with the U.D.F., feel acutely the tragic loss of a congenial friend, whose constant good nature, generosity and thought for others were such marked features of his daily life and stamped the manner in which he practised his profession. Few have excelled his pleasant and even temperament. His enthusiasm for the special branch of medicine and surgery in which he worked was always stimulating. His honesty and integrity in all matters was absolute. We salute a modest, kindly and able professional colleague and friend.

H. B. Stallard, *M.B.E.*, M.D., F.R.C.S.
London
18 March 1950

AN UNUSUAL FINDING IN SPHENOIDITIS

To the Editor: A detailed investigation of the Sphenoidal sinus for signs of disease includes cytologic study of the aspirated secretions. Various authors have commented on their findings. Tillotson, in a study of 25 sphenoid sinuses, found a preponderance of mononuclears in chronic infections, and polymorphonuclear predominance in the acute stage of infection. He makes no mention of the presence of eosinophils.

Sewall, in a study of 98 specimens from 65 patients, found polymorphs, mononuclears and lymphocytes in various combinations. He stressed the absence of eosinophils.

Peele and Le Jeune studied the aspirated sphenoidal contents in 75 cases in 38 of which chronic suppuration was present; in over 50% of the cases a definite allergic factor was present.

These authors state: 'We rarely found eosinophils in the pus from a sphenoid lavage. We consider this to be a characteristic feature of the cytology of sphenoiditis.'

The writer, in the course of the routine investigation of a case of chronic asthma of some 15-20 years' duration, aspirated the contents of the left sphenoidal sinus. Examination of the material yielded the following cytological information: polymorphs, 21%; lymphocytes, 9%; eosinophils, 70%. The culture was sterile.

This case is the only one among the writer's series to show eosinophils. In view of the unusual nature of this finding the matter was thought worthy of report.

The writer wishes to thank Drs. M. H. Finlayson and H. W. Clegg who undertook the bacteriological and cytological investigation.

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M. Glass.
620 Boston House,
Strand Street,
Cape Town.
22 March 1950.